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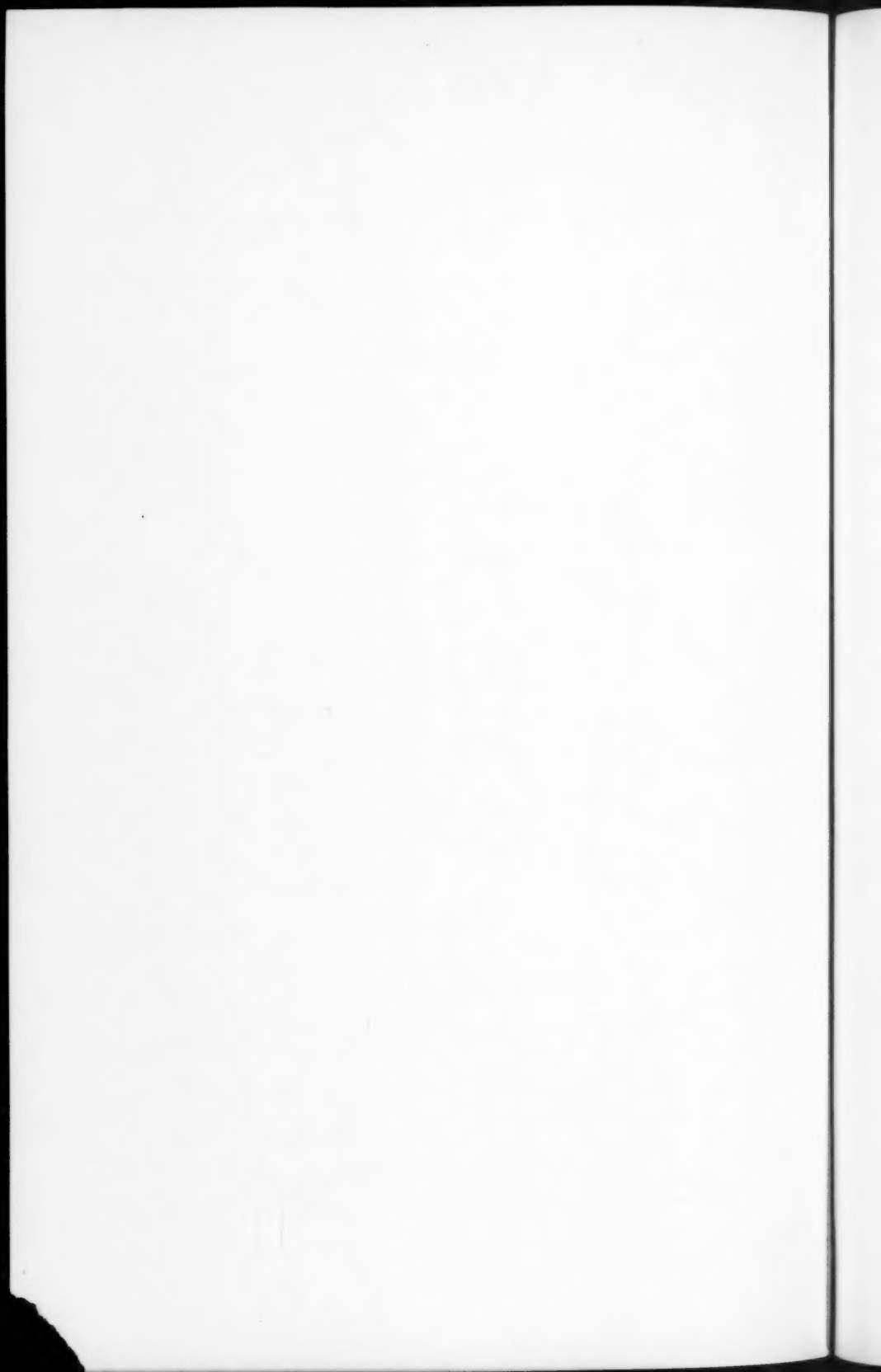
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ANNALS
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XXII

COLLAGEN DISEASES
OF THE
UPPER RESPIRATORY TRACT

M. ARSLAN, M.D.

PADUA, ITALY

This paper is an attempt, the first of its kind in all literature on the subject, to provide a uniform setting for all the aspects of the problem and must therefore first of all clearly illustrate the premises we have followed.

It seems superfluous to point out that whatever conclusions or postulates we may reach, they should not be taken as permanently established; the uncertainty of our information on the pathology of collagen, the polymorphism of the better known clinical pictures of collagen diseases, the fluid nature of nosologic identification, the inconstancy of the semeiologic, humoral and other signs which are considered to be specific of said diseases, and finally, the divergence of opinion on the part of many experts on this subject, all these various factors restrict our work to the mere stating of the starting points for a study and understanding of the subject. This reservation is particularly necessary in our case, since collagen diseases pertaining to otolaryngology can only be circumscribed manifestations of an affection of the collagen itself, which may, or may not, constitute the first stage of a generalized clinical picture of collagen disease. We say "clinical" and not "morphological" advisedly, since it is a known fact (which we ourselves have been able to observe) that there do

From the Clinic of Oto-Rhino-Laryngology, Padua University (Prof. M. Arslan, M.D., Director). Read before the Sixth International Congress of Otolaryngology, Washington, D.C., May, 1957.

exist collagen diseases which are limited to the upper respiratory tract, without any subjective or objective symptoms in the viscera or in the great organic systems, but in which humoral, bioptic and other signs clearly show that a generalized alteration of the collagen is in progress.

The first distinction which we must make in the group of otolaryngological manifestations of collagen diseases is between those which become evident in the course of a generalized collagen disease (and which we will term ENT *secondary* manifestations) and the *primary* or *circumscribed* affections mentioned above.

The secondary manifestations comprise those clinical pictures, and sometimes more simply those morphologic alterations or functional disturbances which can be observed, e.g., in the course of disseminated lupus erythematosus, of malignant dermatovisceritis, of diffused scleroderma, etc. (we have not mentioned panarteritis nodosa because the latter disease plays a special role in our assumption, to which we shall revert in the following pages).

We shall dispense here with describing these secondary manifestations (whose various aspects have often been illustrated in the literature in connection with descriptions of the objectivity of clinical cases of various collagen diseases) for the reason that these secondary manifestations are often not a clinical expression of an affection of the collagen connective tissue of the upper respiratory tract or of the ear, but are aspecific lesions.

The study of primary forms of collagen disease of the otolaryngological sphere is very much more interesting and important.

We refer to those forms whose etiology is unknown and which present clinical, morphological and histopathological features identical with or similar to those of the systemic collagen disease, which authors call pathognomonic (even within the limits imposed by our incomplete knowledge of the subject).

If for such a nosologic classification we applied the criteria suggested by several authors, and particularly by Malaguzzi Valeri, for defining generalized collagen disease, we should have to consider a large number of affections of the upper respiratory and digestive tracts and of the ear, which are characterized by the following features: 1) the extremely chronic nature of the morbid picture; 2) its evolution in recurrent "flares"; 3) its character as an "automaintain-

ing" disease; 4) evidence of an attenuated infective etiology; 5) histopathologic aspect of a chronic granulomatosis with obvious signs of an involvement of the collagen connective tissue; 6) almost always, signs of disreactivity, whether expressed in the histopathologic patterns of the lesion or whether, but more rarely, by the hematologic signs, and so forth.

It can be safely stated that for the otologist there are innumerable clinical instances of ulcerative, proliferative, desquamative, edematous, or dystrophic lesions, and lesions caused by an endocrine disorder, of which today we know nothing, either as regards their etiologic agents or their pathogenetic mechanism. I hope I will be justified in trying to classify these affections as collagen diseases or, in a broader sense, as mesenchymal diseases.

But it is obvious that a policy of prudence and cautiousness must necessarily be adopted: this is the warning given by all experts on the problem, from Klemperer to Cavallero.

We shall now rapidly survey a few aspects of the problem, mentioning particularly the contributions which our Department of Otolaryngology has made to the study of otolaryngological collagen diseases. Our investigations covered the etiology, the histopathology, the semeiology, the clinic picture, the treatment, of the otolaryngological collagen diseases. We have endeavored to maintain constant co-ordination among the various authors working in our Department so that the investigation should follow a uniform general plan. Thus we succeeded in applying a new semeiologic method, the arteriography of the external carotid, to the diagnosis of collagen diseases, after the histopathologist had discovered signs of particular vascular involvements in the proven collagen disease cases. The same co-operation was achieved for therapeutic applications.

ETIOLOGICAL ASPECTS

As regards the factor connected with mesenchymal disreactivity, according to Lunedei's meaning, and intended as a morbid condition favoring, if not substantiating, the onset of a collagen disease, Fioretti has evidenced (1953 and following years) the various patterns of the allergic-hyperergic historeactivity of the upper respiratory and digestive mucosa, differentiated according to the various segments of the mucosa, by means of the experimental device for the introduction of antigen, by the centrifugal carotid means, into previously sensitized animals.

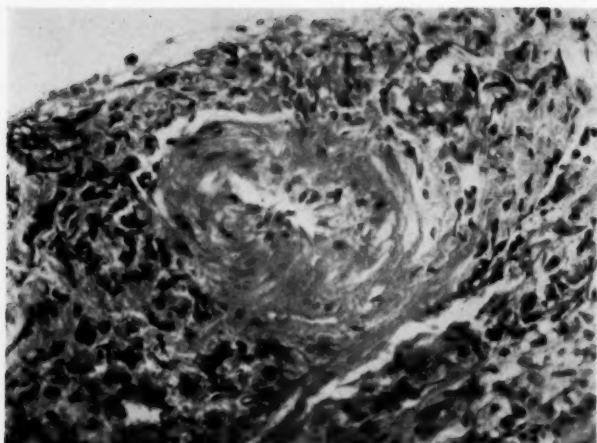


Fig. 1.—Photomicrograph of a severe vascular lesion in granuloma gangrenescens. The vasa lumen is nearly completely occluded by the endothelial proliferation. In the media homogenization of muscle fibers is noted. The adventitia is invaded by granulation tissue.

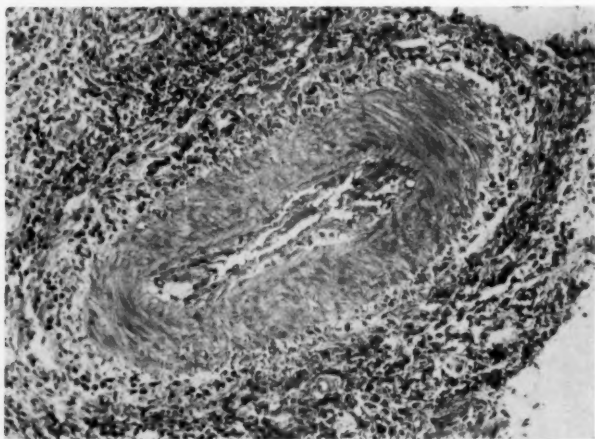


Fig. 2.—Another aspect of severe arteriosclerotic lesion in granuloma gangrenescens. Note the intima detached from the wall and swollen, the edema of the media and the adventitial proliferation.

He proved, among other things, that the tonsils play no part in these historeactivity phenomena and he further evidenced the distribution of the more intense allergic-hyperergic phlogosis areas according to differences in the epithelium, to the vessels situation, and so on.

Fioretti's research is part of a more general investigation of the physiopathology of the tonsils which has been continuing for many years in our Department: now the assumptions deriving from this investigation seem to agree exactly with the recent hypothesis formulated by Chini and Malaguzzi Valeri, concerning the relationship between focal infections and collagen diseases, and the possibility that both groups of diseases are "auto-antibody diseases."

In fact, according to Fioretti's histopathologic and experimental research, the histofunctional processes which take place in the nodular parenchyma of the tonsils appear to be the most likely to form "auto-antibodies."

This convergence of several postulates, as the result of research work carried out by different authors, has clinical conformation in the affinity (sometimes identity) of focal infections and collagen diseases.

HISTOPATHOLOGICAL ASPECTS

The histological research carried out on the otorhinolaryngological affections which were suspected of being collagen diseases was directed towards the twofold object of proving that the mesenchyma plays a role in the disease, and also, if need be, that widespread lesions of the collagen are present (V. Ricci).

The histological techniques employed were those which would best show the specific alterations of the collagen, such as fibrinoid degeneration, modifications of the mucopolysaccharides, and alterations of the fibrillar and precollagen structure.

In order to prove the presence of widespread alterations of the collagen of the system of small vessels, we resorted to muscle biopsy, carried out on the flexor surface of the upper and lower limbs.

A) The "*Granuloma Gangrenescens*" or midline granuloma seems to be the otorhinolaryngological disease which has all the clinical, anatomic and pathological features necessary to consider it a

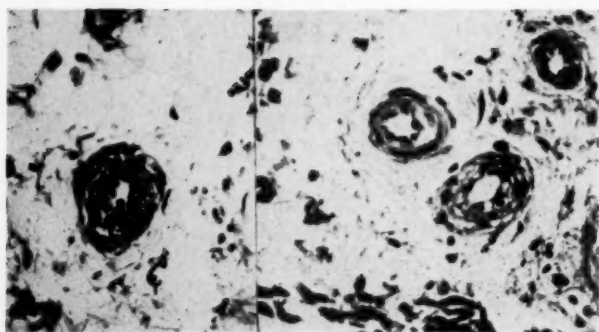


Fig. 3.—In the beginning granuloma gangrenescens it is possible a tendency to swelling and inhibition of the media in the arteries of small caliber, with hyperplasia of the intima.

widespread collagen disease, closely connected with the group of vascular processes headed by panarteritis nodosa.

From the literature pertaining to this field that we consulted we are citing only the work of Williams, Hagens et al., and Fahey et al., to whom must go the credit for having demonstrated the relationship between this pathological entity and vascular collagenous lesions.

In the cases we studied, although the nasal lesion showed a quite serious morbid picture, it was isolated and was not accompanied by any clinically noticeable lesions in other organs. In particular, no functional lesions were ever found in the kidneys.

The histological examination of these lesions shows a lively proliferation of reticular-histiocyte cells. These are mainly polyhedral and ovoid in shape, divided by thin argyrophyle walls, which thicken here and there into hyaline trabeculae. The nuclei are fairly regular in size and susceptible to staining: occasionally cells are found with a highly staining lobate nucleus, having a basophile protoplasmic halo. This granulomatous tissue, although reasonably well provided with tiny blood vessels, tends to become necrotic through coagulation.

Deep seated lesions of the small and medium arteries are particularly interesting.

These blood vessels present an irregular swelling and proliferation of the intima, which is often detached from the wall. In the space thus formed between the intima and the media, an amorphous, slightly basophile substance collects, which can be stained cardinal red by the Hotchkiss-MacManus method, reddish purple with toluidin blue, and blue green by the Rinheart-Abul-Hai method. In the small arteries, the proliferation of the intima occasionally even occludes the lumen (Figs. 1,2,3).

The media of the small arteries presents a homogeneous, structureless, thickened appearance. The Van Gieson method stains it a yellow-orange; the Azan-Mallory method, a homogeneous red; while toluidin blue causes slight and irregular metachromasia. In the median arteries the media is less altered: here, too, however, it is easy to find homogenizing foci of the muscular fibro-cells, especially in the inner part of the wall, as well as an initial connective tissue replacement.

The inner elastic limitans in all the arteries presents different alterations: it is sometimes in fragments, or more or less extensively destroyed, and sometimes irregularly thickened or doubled.

In the adventitia, besides a swelling of the collagen, which presents a fibrinoid aspect, a lively and irregular proliferation of the fibroblasts and histiocytes is found.

To conclude, we find in these forms, alongside the granulomatous lesions, evidence of a considerable obliterating connective-elastic endoarterial disease, which suggests a relationship between the granuloma gangrenescens and the Buerger type obliterative arterial disease.

The histological examination of fragments of the muscular masses and of the subcutaneous tissue from the upper and lower limbs showed some irregularity in the size and susceptibility to staining of the muscle fibers. Alongside perfectly normal elements, others are found which are swollen, hardly susceptible to staining, with faint or even invisible striae. In some fragments a numerical increase of the sarcolemma nuclei is found. The arterioles and the intramuscular capillaries present a definite thickening of the intima which by the Hotchkiss-MacManus staining method appears as a small cardinal red border. The nuclei of the intima are globose and protrude in the lumen. Around the tiny vessels small groups of lympho-monocyte elements are frequently found. The infiltration is fairly widespread but never becomes a real nodule.

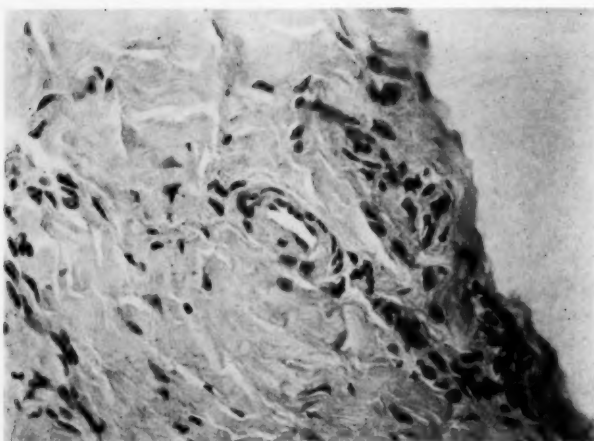


Fig. 4.—In the Gougerot-Sjögren syndrome the sclerhyaline alteration of the submucous connective tissue is evident: this latter forms great homogeneous clods, especially around the tiny arterioles.

These muscular lesions are very interesting because they complete laboratory research and make it possible to classify the affection among the collagen diseases and to assign it to the panarteritis nodosa group. It is, in fact, commonly known that in this disease the lesions are usually very widespread in the muscle tissue, so that biopsies carried out on muscles showing no clinical signs of a lesion very often give positive results. In this connection Barbera and Malaguzzi Valeri have pointed out the significance of non-specific cellular infiltrates and regressive and degenerative processes in the muscle fibers.

B) The *Gougerot-Sjögren Syndrome*: the histological examination of the mucosa has evidenced, alongside the atrophy of the epithelium mucosa which appears to be flattened and desquamated, a considerable thickening of the underlying connective tissue. This is thickened in big hyaline trabeculae, which are sometimes fragmented in lumps (Fig. 4). The small blood vessels present thickened and sclerotic walls; occasionally there are accumulations of phlogistic elements in the interstice and in the adventitia of the blood vessels.

The muscle biopsy has shown a number of lesions very similar to those already described in the *granuloma gangrenescens*, although less marked.

C) *The Collagen Disease of the Auricular and Nasal Cartilages*:* we observe the presence in the perichondrium of nests of phlogistic elements of a granulomatose nature which are deep seated and disrupt the cartilaginous tissue. Staining reveals a fibrinoid degeneration of the connective tissue around the phlogistic nests.

The cartilage, which is infiltrated and dissociated by connective tissue, presents considerable regressive alteration of the cells with pyknosis and cytolysis. There is also a fractioning of the elastic cartilage fibers which appear to be transformed into large and small granules.

The periadventitial space of the blood vessels presents edema and is often occupied by a "sleeve," sometimes thin, sometimes clearly evident, of inflammatory cells, among which plasmacellular elements prevail.

D) *Amyloidosis of the Larynx*: the histological and histochemical examinations have shown, besides the atypical staining, characters of the amyloid substance, more or less extensive granulomatous formations, with the participation of tiny vessels and, sometimes, with the presence of fibrinoid necrosis.

E) *In Ozena and in Allergic Ethmoidal Polyposis* we have never found histological elements comparable with those described above, in which the characteristic lesions of a collagen disease could be observed. Muscle biopsy has always given positive results.

F) *Severe Focal Infections*: muscle biopsy was positive, with lesions comparable to those previously described (Fig. 5).

SEMEIOLOGIC ASPECT

The fundamental importance of this aspect of the problem is self-evident. We should, in fact, establish now what clinical, humoral, bioptic and other signs should be accepted as surely indicating a collagen disease; and we should further establish within what limits, and with what reservations or exceptions, those signs should be evaluated.

* It is due to D. Megighian that we have the illustration of a highly demonstrative case of collagenosis of the nasal and auricular cartilages in a subject affected with a severe rheumatoid arthritis, extended also to the cricoarthritis articulations.



Fig. 5.—Histological aspect of a fragment of muscular tissue in a case of focal disease. Note the small accumulations of lymphocytes around the blood-vessels, the augmentation of sarcolemmatic nuclei and the alterations in form and in volume of the muscular fibers.

It is now time for us to state which are the otolaryngological affections to which we have directed our attention, qualifying them as real or supposed collagen diseases. *We wish to repeat that in an introductory study of the problem we are compelled to make the scope of our presumptions and indications as broad as possible.*

In a preliminary note¹ and in a subsequent note by Sala,¹² six diseases were brought to the notice of scholars: 1) *granuloma gangrenescens* (the "midline granuloma" of American authors and the "granuloma malin" of French); 2) *amyloidosis*; 3) *sarcoidosis*; 4) *scleroderma*; 5) *typical allergic ethmoidal polyposis*, i.e., without inflammatory signs; 6) *ozena*. Now, after thoroughly analyzing the various aspects of the problem, we believe that sarcoidosis (or Boeck's sarcoid) should be excluded from that list, while three other morbid manifestations of the otorhinolaryngeal region should be added to it: 1) *Gougerot-Sjögren syndrome*; 2) *the collagen disease of the auricle and nasal cartilages*, which is a primary or secondary form of rheumatoid arthritis; 3) *focal infection*.

Many authors believe that dysproteinemia with hyper-gammaglobulinemia is always present in collagen diseases: however, Cavalero affirms that it is far from being constant and typical.

Our systematic search for the electrophoretic outline in cases of affections included in the above lists has shown pathological hypergamma-globulinemia values only in the following diseases: granuloma gangrenescens, Gougerot-Sjögren's syndrome, and some cases of focal infections.

In all cases of granuloma gangrenescens, of Gougerot-Sjögren's syndrome, of a collagen disease of the auricle and nose cartilages, muscle biopsy has given positive results, revealing the histopathological patterns which were amply illustrated in the previous part of this paper.

As stated above, the finding of histological pictures showing vascular involvement in cases of granuloma gangrenescens justified a comparison between that disease and obliterating arterial disease of Buerger's type.

Taking into account the diagnostic difficulties which the granuloma gangrenescens presents particularly in the initial stages, we considered the possibility of ascertaining, through arteriographic examination, the state of the arterial canalization in the nose and sinus region, which is the seat of the affection. In Figure 6 is shown an arteriogram of the external carotid of a patient with a rhino-sinus-pharyngeal granuloma gangrenescens (Bötner).

The corresponding arteriogram shows the smallness of the area of arteries tributary to the external carotid. This can be seen particularly at the level of the dural vascular bed, where the absence of several superimposed planes affords a better ground for judging the size and abundance of the arterial network. But the most important fact in the arteriogram is supplied by observing the internal maxillary artery. The latter, at its distal end, instead of presenting the terminal branches it usually has after its entrance into the pterygopalatine fossa, shows a progressive and rapid reduction of its lumen, so that it has a tapering or "mouse tail" appearance. Radiologists are familiar with this aspect, which is typical of limb arteriograms of patients suffering from Buerger's disease.

No injected ramifications are visible distally to the region described, and this region corresponds to the seat of the granuloma gangrenescens in this patient.

THERAPEUTIC ASPECTS

Besides medical treatment based principally on steroid hormones on which O. Scala has carried out original research in our Department,

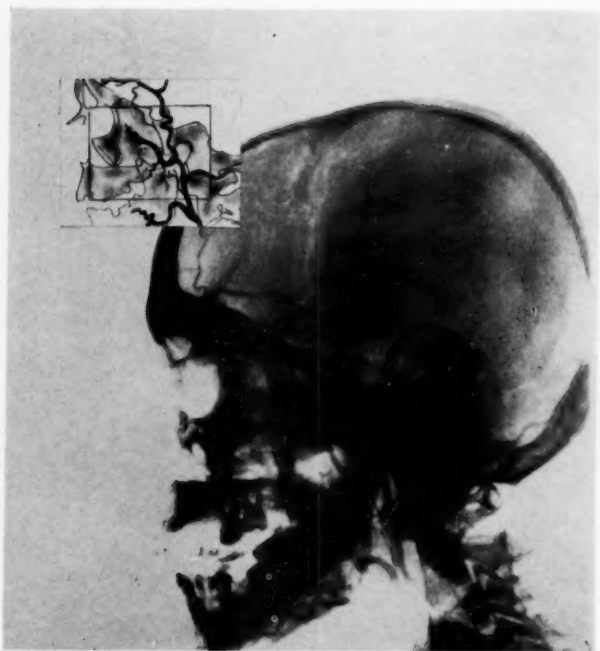


Fig. 6.—Arteriogram of the external carotid in a case of granuloma gangrenescens. Note the smallness of all the vessels, and the characteristic "mouse-tail" appearance of the internal maxillary artery.

we have employed a new surgical therapy for granuloma gangrenescens. The idea of this therapy sprang directly from the findings of the histopathological survey of our granuloma cases, which we have reported in detail and which, as we have seen, have been confirmed by the arteriographic test. This third link in the chain consists in applying to the granuloma gangrenescens the same surgical therapy which has been employed successfully for some time in Buerger's disease of the limbs, that is to say, in a morbid state which has much in common with panarteritis nodosa and therefore with granuloma gangrenescens. The latter in fact is an initial circumscribed manifestation of panarteritis nodosa. It is common knowledge that this therapy consists of the interruption of the sympathetic innervation of the affected part.

In two of our granuloma gangrenescens cases, which were entirely typical as regards the histopathological, hematologic results from the muscle biopsy, cervical sympathectomy was, in fact, carried out on the side where the destructive lesions caused by the granuloma gangrenescens were largest. In one of these two cases the operation has been performed by G. Pagnini, chief otologist of the City Hospital of Pesaro.

In both cases cervical sympathectomy has led to a complete cicatrization of the ulcerative necrotic lesion and to complete recovery, which has been continuing for years. It should be kept in mind that granuloma gangrenescens almost invariably has a lethal course. Our two cases, cured by the surgical treatment described above, therefore fully deserve particular emphasis and attention.

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BIBLIOGRAPHY

1. Arslan, M.: Collagen Disorders in Otorhinolaryngology. *Minerva O.R.L.* 4:1, 1954.
2. Arslan, M.: Collagen Disorders in Otorhinolaryngology. 45th Congr. Soc. It. O.R.L., Pisa (Sept.) 1957.
3. Bötner, V.: Arteriography of External Carotid in Collagen Disorders. 45th Congr. Soc. Ital. O.R.L., Pisa (Sept.) 1957.
4. Fahey, J. L., and Coll.: Wegener's Granulomatosis. *Am. J. Med.* 17:168, 1954.
5. Fioretti, A.: Experimental Focal Diseases. 45th Congr. Soc. It. O.R.L., Pisa (Sept.) 1957.
6. Hagens, E. W., and Coll.: ACTH in Lethal Granuloma. *Arch. Otolar.* 57:561, 1953.
7. Malaguzzi Valeri, C.: Collagen Diseases. Roma, 1956.
8. Megighian, D.: Collagen Disorders of Aural Cartilage, etc. *Arch. It. O.R.L., Suppl.* 26, 1955.
9. Megighian, D.: Cervical Sympathectomy in Collagen Disorders. 45th Congr. Soc. It. O.R.L., Pisa (Sept.) 1957.
10. Ricci, V.: Histology of Collagen Disorders in O.R.L. 45th Congr. Soc. It. O.R.L., Pisa (Sept.) 1957.
11. Ricci, V.: Granuloma Gangrenaescens and Collagen Diseases. *Minerva O.R.L.* 8:3, 1958.
12. Sala, O.: Therapeutics in Collagen Diseases. 45th Congr. Soc. It. O.R.L., Pisa (Sept.) 1957.
13. Wegener, F.: *Beitr. Z. path. Anat. u. allg. Path.* 10:35, 1939.
14. Williams, H. L., and Hochfilzer, J. J.: Effect of Cortisone on Idiopathic Granuloma. *ANNALS OF OTOLARYNGOLOGY AND LARYNGOLOGY* 59:518, 1950.

XXIII

THE GROUND SUBSTANCE OF THE NASAL TURBINATES

A PRELIMINARY REPORT OF HISTOCHEMICAL STUDIES

ALEX WEISSKOPF, M.D.

AND

HELEN F. BURN, M.S.

SAN MATEO, CALIF.

Histochemical studies of the ground substance of connective tissue are playing an increasingly important role in the understanding of its physiology, both normal and pathological. The following is a preliminary report of the background of material and staining techniques involved in this study as related to the nasal mucous membranes. The philosophy of this study conceives the intercellular substances as not merely a packing and protective material, but as an important physiologically active substance which takes a primary place in the activities of the tissue. There has been an enormous wealth of literature, conferences, and books on the subject of connective tissue since Duran-Reynals'¹ classical article on the Spreading Factor appeared in 1928. It would be impossible in an article of this nature to attempt to even partially cover the immense literature in this field. Particularly useful for a broad understanding of this field are the works edited by Asboe-Hansen,² Miner,³ the Ciba Foundation Symposium,⁴ and Tunbridge.⁵ These contain a very extensive bibliography of the original work. In the interest of space and time only the prime reference articles will be mentioned in this paper since they offer an excellent review of an entire branch of this study.

There are still many unanswered questions regarding the basic mechanism of nasal physiology. In the past the mechanistic approach has been helpful in our understanding of nasal physiology. Proetz, Hilding, and others have laid proper emphasis on the protective and cleansing ciliary action of the nose. The antibacterial and immu-

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nologic aspects have been studied, but there have been few means of attacking the basic problem of nasal changes as seen in allergy, polyposis, and vasomotor rhinitis of whatever etiology. It is quite evident that there must be other factors than vascular to cause engorgement of the nasal tissues. The question arises: what is the nature of the "edema" in such a variety of conditions as nasal congestion due to hormonal changes (pregnancy, thyroid), allergy, and emotional stress? This study is an attempt to answer this question.

Duran-Reynals⁶ stressed the unity of all physiological processes. He points out that sooner or later all reactions must involve the ground substance. Hormones, metabolites, toxins, food substances, and bacteria must all traverse the ground substance to reach the cells. All inflammatory reactions occur in the connective tissue and therefore are intimately related to the ground substance. Karl Meyer⁷ in 1947 presented a comprehensive review of hyaluronic acid and hyaluronidase. Subsequently⁸ in 1957 he reviewed the chemistry of the known ground substances. The most important and best known constituents are the acid mucopolysaccharides, especially hyaluronic acid and chondroitin sulfate A, B, and C. Of great interest to the study of nasal physiology has been the close association of histamine and heparin to these substances. The acid mucopolysaccharides are long polymers of very high molecular weight in the neighborhood of eight million.

Hyaluronidase is the original spreading factor described by Duran-Reynals. It is an enzyme acting specifically on these mucopolysaccharides. Hyaluronidase is obtained from two sources. The animal origin is primarily from testicular extraction and is the commercial hyaluronidase used clinically. A lesser known animal source is snake venom. The second group of hyaluronidase is obtained from bacterial culture, primarily the pneumococci, streptococci, staphylococci, and clostridium Welchii. Hyaluronic acid is broken down to the disaccharides, N-acetylated glucosamine, and glucuronic acid by depolymerization. Of more importance than the actual disaccharides that are formed is the marked physical changes that occur in the properties of the mucopolysaccharides upon alteration in their polymer size. The polymer size is of great physical-chemical importance. At very low concentrations it has great water binding capacity. Its viscosity acts as a barrier with an active control of the transport of metabolites, toxins, hormones, and other elements found in the interchange between blood vessels and tissue cells.

Persson⁹ in 1953 postulated the following functions of the ground substance:

1. Storing and transporting of water and electrolytes.
2. A shock absorber and lubricator.
3. Mechanical barrier against the spread of foreign substances and bacteria.
4. Connective tissue repair.
5. An end-organ for hormones, including thyroid, sex, and the adrenocortical.

He points out that the functional state of the matrix depends upon its gel state. This varies markedly from moment to moment, depending upon many factors as yet unknown.

The presence of minute amounts of hyaluronidase is postulated. This would disappear almost as soon as it was formed, having exerted its effect on the mucopolysaccharide substrate. Mayer and Kull¹⁰ found that there are small amounts of free hyaluronidase in the skin. They felt that this was an integral part of the hyaluronic acid formation-decomposition system and may explain some of the pathologic processes seen with inflammation, allergic as well as infectious and chemical. It is interesting to note that in severe dermatitis and burns of guinea pigs skin there is an increase of forty times the normal value of free hyaluronidase. Hechter¹¹ points out that there are other spreading factors besides hyaluronidase. These include the azoproteins, ascorbic acid, and other reducing agents. Conversely, there are direct inhibitors of the spreading action of hyaluronidase (Dorfman¹²). Others have no direct action on the hyaluronidase itself but act upon the tissues. These substances include epinephrine, morphine, the antihistamines, salicylates, rutin, and adrenocortical extracts. These act indirectly by preventing edema, reducing the blood flow, neutralizing toxic actions, or decreasing capillary permeability.

A great wealth of material has arisen on the action of hyaluronidase on the mucopolysaccharides of the ground substance. The primary change is depolymerization with subsequent loss of the binding gel-like state. McMaster and Parson¹³ in 1950 beautifully displayed the action of hyaluronidase on the blood vessel wall. By injecting various isotonic dyes with hyaluronidase into the lymphatics of the ear of a mouse the breakdown of the intercellular cement could be followed. It was found that the dye extruded from the lymphatics in tiny hair-like bands which could not be dislodged. They postulated that the dye extravasates along tiny fibrils in the connective

tissue matrix and that this is the normal state of fluid in the tissue spaces, much as a film caught between two plates of glass.

Of special interest in the study of the nose is the work of Mayer and Kull¹⁰ who found the antihistamines pyribenzamine and antistine possess definite antispreading factor action. They found that hyaluronidase is not only a spreading factor for such inert agents as India ink, but also increases the allergic reaction of the skin of sensitized animals probably by permitting increased spread of toxins and inflammatory agents. The spreading action and the increased sensitization due to hyaluronidase was markedly decreased when the antihistamines were first given. It must be pointed out that Fischel¹⁴ could find no significant effect of the antihistamines on the necrotizing bacterial allergy of the tuberculin type or on the Arthus' phenomenon. The salicylates seemed to prevent some of the local edema but likewise did not prevent the Arthus reaction.

There have been many studies on infection and the effect of hyaluronidase on its spread. This was the original premise of Duran-Reynals. A very tempting mechanism has been postulated in which a correlation between hyaluronidase production and virulence could be made. A bacteria producing considerable amounts of spreading factor would theoretically be able to penetrate more rapidly and invade the tissues before the protective mechanisms are able to function. Sallman and Birkeland¹⁵ present evidence for the correlation between the pathogenicity of hemolytic streptococci and the amount of hyaluronidase produced by this strain. If hyaluronidase is added to a strain of non-hyaluronidase producing streptococci the virulence was increased many fold. Conversely, if the hyaluronidase was removed from a hyaluronidase-producing strain there was a decreased virulence. They were able to decrease the mortality of chick embryos significantly by adding sodium salicylate. They theorized that genisic acid, an oxidation product of salicylate, united with the hyaluronidase and thus prevented its activity. It must be emphasized that many pathogenic organisms are encapsulated. This capsule is hyaluronic acid. These organisms produce very little hyaluronidase. This is a subject of great interest and should be investigated further.

Pike¹⁶ could find no relationship between the production of hyaluronidase and virulence of a strain of group A beta hemolytic streptococci in the mouse. It must be pointed out that the spreading factor is a two-edged sword. It helps the invading organism enter the host tissues. On the other hand, it aids the host by diluting the invading bacteria below the critical survival point. This has been

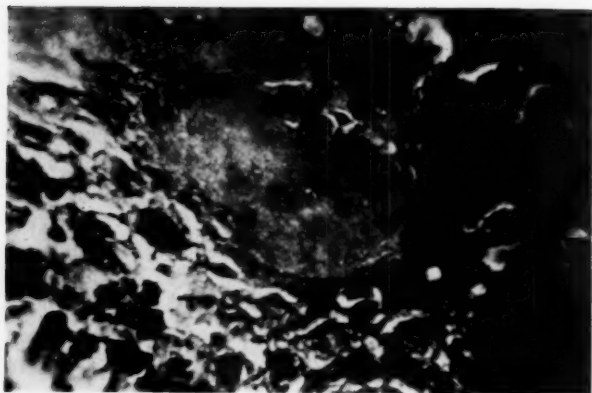


Fig. 1.—Colloidal iron stain, 970X magnification. Ground glass appearing basement membrane is visible. The darker staining material is blue acid mucopolysaccharide. The lighter is collagenous material.

called the "colonial effect." The bacteria are compared to a group of colonists in a new land. If the colonists spread themselves out too thinly, their chance for survival is decreased, whereas if they maintain themselves as a small rigid unit the ability of the colony to survive improves.

There are many other factors involving the ground substance which are known to play a role in nasal physiology. It is well known that various hormones will cause marked changes in the status of the ground substance. Interestingly, nasal congestion is influenced by many of these hormones. Asboe-Hansen¹⁷ points out that the intercellular and interfibrillar ground substance of the skin is mainly hyaluronic acid with some chondroitin sulfuric acid. In his study of 26 myxedematous hypothyroid cases he found a marked increase in the amount of ground substance and number of mast cells. He theorized that this was the cause of myxedema. It has been postulated that the excessive ground substance in myxedema is due to an overactivity of the anterior pituitary gland. Proud¹⁸ speculates on the mechanism of the changes in the turbinates of thyroidectomized dogs. He notes "swelling and separation of the collagen fibers." Histochemical studies on this material should be most instructive.

Iversen¹⁹ presents a detailed report of other hormonal influences on connective tissue. The classical example of cockscomb growth

following testosterone and the macacus monkey sex skin due to estrogen are excellent examples of the overproduction of ground substance due to hormones. Is pregnancy vasomotor rhinitis related to this?

The corticoids have created a great deal of interest. Their clinical action is better known than the mediator of the action. It is known that the corticoids decrease all the connective tissue elements, especially the number and size of mast cells and fibroblasts. The corticoids indirectly decrease the spreading effect of hyaluronidase. Rawlins²⁰ in 1956 gave a review of the corticosteroids as related to otolaryngology.

There is a striking similarity in the reactions of the ground substance as reviewed above with many of the changes seen in the nasal turbinates. This appears to be more than coincidental. Our program was set up to utilize the histochemical stains for further study of nasal physiology as seen in normal and pathological states. The histochemical stains are erratic and somewhat difficult to work with. A considerable amount of time was spent on the staining procedure. Fresh post-mortem material was used and stained repeatedly to ascertain the reliability of the staining procedure. This is most important for minor variations in technique will caused marked variation in the final histological picture. To prove the presence of acid mucopolysaccharides one of the series of each tissue was incubated in hyaluronidase, both bacterial and testicular. Loss of staining characteristics after bacterial hyaluronidase is presumptive evidence that the substance is hyaluronic acid alone. After testicular hyaluronidase extraction, loss of staining characteristics would indicate the presence of hyaluronic acid and all the chondroitin sulfuric acids except chondroitin sulfate B, which is presumed to be heparin. This reacts to none of the hyaluronidase.

HISTOCHEMICAL TECHNIQUE

Small pieces of tissue were fixed in 10 per cent neutral formalin, dehydrated, cleared in chloroform, and embedded in 52° to 54° C tissue mat. The time for these operations was kept as short as possible. Serial sections were cut at a thickness of 5 to 7 micra. The water in the spreading bath was kept at a temperature of 45° to 57° C and each section was floated and quickly drawn up on an albuminized slide. Mayer's albumin was made in a proportion of 40 ml of glycerine to 60 ml of fresh albumin to enhance the adhesive quality. The section was drained for a few minutes and then covered with a piece of wet

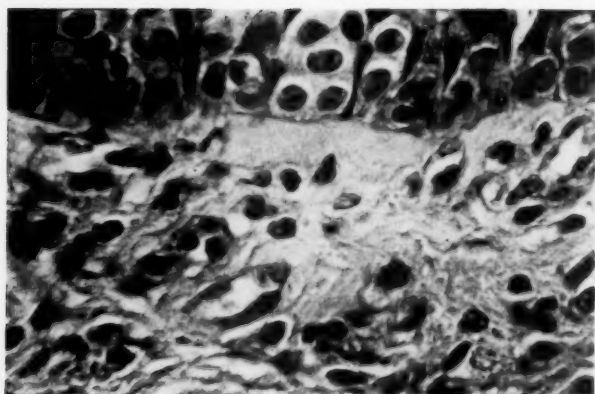


Fig. 2.—Colloidal iron stain, 970X magnification, with testicular hyaluronidase. There is absence of dark staining mucopolysaccharide in basement membrane and lamina propria. Note persistence of dark staining material, probably mucus, in and between the epithelial cells.

bibulous filter paper and rolled with a photographic roller to remove possible air bubbles. The slides were air dried for 24 hours before being placed on a hot plate for one hour. Mucopolysaccharides are more soluble in hot water and care must be taken to keep the contact with it as short as possible.

The sections were deparaffinized with xylol, hydrated, and rinsed twice with distilled water. One of each series was stained with Harris' hematoxylin and eosin Y for a general histological and cytological study.

For each of the three stains described below, three consecutive sections were used. The first was untreated, the second was treated with testicular hyaluronidase, and the third with streptococcal hyaluronidase. The enzyme extraction and staining were carried out on the same working day to avoid possible change in the mucopolysaccharides or loss of sections. A section of umbilical cord was included with each set of stains as a test. Since it has a high concentration of hyaluronic acid, it reflects the extent of enzyme extraction and the staining technique. All glassware used was chemically clean. McIlvaine-Lillie buffers were used throughout.

Wydase® (Wyeth) was used as the source for testicular hyaluronidase. Five hundred TRU was dissolved in 1 ml of buffered normal saline, pH 6.0, and added to 25 ml of buffered saline for each five slides. The streptococcal hyaluronidase was furnished in a 500 mg quantity. This was dissolved in 45 ml of buffered saline, pH 6.0, and stored in 3 ml units in the deep freeze. One ml of this solution was used per 25 ml of buffered saline for five slides, giving a concentration of 11+ mg per jar.

The slides were placed in Coplin staining jars containing 25 ml of buffered saline and placed in a water bath at 37° C for one hour. Then the hyaluronidases were added to the respective jars and they were all incubated at 37° C for 90 minutes. During that time, they were agitated every five minutes. Thus the untreated sections were exposed to the same experimental conditions of saline and heat as the treated sections. The slides were washed in running water for 30 minutes and rinsed in two changes of distilled water.

Several stains were tested and discarded. These included azure-eosin,²¹ HCl-orange G-methyl blue,²² Alcian blue (Steedman's²³), Lison's,²⁴ Mowry's,²⁵ and Kramer and Windrum's azure A.²⁶ The following were finally selected for this study:

1. Gomori's toluidine blue.²⁸
2. Rinehart and Abul-Haj's modification of Hale's colloidal iron.²⁷
3. Periodic acid-Schiff.²⁹

For each stain used, the three slides, treated and untreated, were carried through together to prevent slight differences in strength or timing. All stains were discarded at the end of the day.

Several samples of toluidine blue made by the National Aniline Company were tested, one which produced the most metachromasia was selected, and a one per cent aqueous stock solution was made. The sections were placed in a pH 5.5 distilled water rinse for two hours. The staining solution was made with 0.5 ml of filtered stock solution and 49.5 ml of distilled water. It was then adjusted to a pH of 5.5. This gives a dye concentration of approximately 3×10^{-4} M. The sections were stained for eight minutes and rinsed with distilled water. The back and sides of the slide were wiped dry, it was dipped into 95 per cent and 100 per cent alcohol, and cleared in xylol. This procedure should demonstrate gamma metachromasia with the beta form changed to the orthochromatic stain.

The colloidal iron stain was carried out as given in the original article except that the slides were washed with running distilled water for five minutes after the iron and the ferrocyanide treatment. No metal forceps were used in handling the slides from the time that they were placed in acetic acid until they were placed in cochineal. The cochineal and the picrofuchsin were filtered before using.

The third stain was alcoholic periodic acid Schiff. The sections were rinsed in 70 per cent alcohol, immersed in periodic acid solution for five minutes, and rinsed again in 70 per cent alcohol. They were placed in Schiff solution in the dark for 20 minutes and washed in running water for 10 minutes. This was followed by filtered celestin blue and orange G as usual. The reducing rinse between the periodic acid and the Schiff solution was omitted when it was found that it reduced the intensity of the staining reaction. The sulfite rinse after the Schiff reaction was replaced by washing and has proved to be sufficient to prevent further staining.

All sections were mounted in Permunt and placed on a hot plate for one hour before labelling.

FINDINGS

The following is seen in normal turbinate tissue taken at biopsy from the anterior tip of the middle turbinate of allergy-free patients. Pontocaine was used for the anesthetic to avoid the shrinking effect of the vasoconstrictors or cocaine.

1. *Toluidine Blue*. This stain depends upon the fact that acid mucopolysaccharides will stain red (metachromasia) rather than the usual blue of other tissue elements. The degree of redness is proportionate to the degree of polymerization. All cells stain with a varying intensity of blue. There is some metachromasia of the mucus in the goblet cells of the ciliated epithelium and in the mucous glands. Mucus is primarily a neutral mucopolysaccharide and is altered very little by the action of hyaluronidase. The connective tissue ground substance generally does not show any evidence of stain. There are faint fibrillar shadows. The basement membrane and the ground substance does not show metachromasia. There are scattered mast cells showing bright metachromatic granules, uniform in size. These cells are found in greatest concentration about blood vessels and in areas of greater fibroblastic activities as evidenced by the number of capillaries and fibroblasts. The greatest metachromasia is seen around and in the blood vessels and the cavernous spaces. Incubation with

testicular enzyme causes complete loss of the metachromasia except for the mast cell granules which persist. These are presumably heparin and so do not react after incubation with enzyme. One finds persistent metachromasia about the blood vessel, although there is a suggestion of slight diminution. This is further evidence that most of the mucopolysaccharides in the blood vessel are of the chondroitin sulfate group.

2. *Colloidal Iron.* This stain depends upon the affinity of the acid mucopolysaccharides for colloidal iron. The iron is then demonstrated by the Prussian blue reaction. The picture is markedly different from the toluidine blue, the basement membrane being now distinctly visible as a band approximately 15 micra in width. There is an impression of a fine ground glass staining blue, most intense towards the epithelial surface. This gradually diminishes until it blends with the mucopolysaccharide of the ground substance of the lamina propria. The intensity of the stain is most varied. The entire picture must be carefully evaluated. There is considerable increase of the blue stain in and about the blood vessels. The mucopolysaccharide can be seen lining the endothelium of the capillaries and forming the intercellular substance between the arterial cells. It is difficult to make out individual cells such as fibroblasts and mast cells because of the densely stained connective tissue ground substance. What appears to be blank on the routine stains now is seen to be filled with the blue mucopolysaccharide and the proteinaceous materials which stain red or bronze in color. Testicular enzyme causes almost complete loss of the blue stain in the basement membrane and lamina propria, revealing a red collagenous background which apparently is the stroma for the mucopolysaccharide matrix. There is little loss of stain from the mucus glands. The bacterial enzyme causes some loss of the blue stain, but a considerable amount is still visible, indicating that most mucopolysaccharides of the turbinate are not of the hyaluronic acid type. The mucopolysaccharides present are in a relatively less polymerized state since they show little metachromasia with the toluidine blue. They are revealed by means of the colloidal iron reaction.

3. *The Schiff Periodic Stain.* This depends upon the splitting of the hydroxyl radical which is oxidized to an aldehyde. This is demonstrated by the leukofuchsin stain, resulting in a vivid pink to red color. This stain is not as specific as the two previous stains, but is a classical stain used in most of the work on connective tissue ground substance. Positive Schiff staining is obtained on glycogen, the mucopolysaccharides, the mucoproteins, glycoproteins, collagen, gelatin,

reticulin, coagulated plasma, and serum. The turbinates show the following: there is the usual intense reaction of the mucus-containing cells which are a very deep violet red. The basement membrane shows a heavy pink line approximately two micra in width lying along the epithelium. Beneath this line is the lighter pink staining ground substance. There is not the distinct separation of the basement membrane from the remainder of the ground substance of the lamina propria as seen with the colloidal iron stain. There is no feeling of separation between the collagenous background and the mucopolysaccharides. However, this stain is excellent for demonstrating the mast cells, whose granules are readily seen as a bright red. Hyaluronidase, both testicular and bacterial, show very little change in the staining characteristics. This would indicate that the Schiff reaction stains the mucopolysaccharide in all stages of polymerization. Since many other substances in the connective tissue are also stained it is difficult to differentiate the mucopolysaccharide from other materials.

In studying these slides it is quite evident that the pitfalls enumerated in the literature on the study of connective tissue of other organs are present in the study of the nasal turbinates. There is considerable variation in the amount of ground substance from one area to the next in the same slide. Cellular elements vary considerably. It is easy to misinterpret the results unless many sections are studied. This may explain the disagreement found in the literature. To avoid the dangers of error due to variation in the stains a rigid system of controls was used. As a final check each batch of stains contained one section of human umbilical cord for comparison. We cannot recommend too highly the use of the umbilical cord, both for the early study of the stains and for control in later series. There are many fine changes in the tissues which can be seen only under oil immersion lens. These can easily be missed except for the fact that they are readily seen in the umbilical cord.

The photographs are of the colloidal iron stain which shows the best differentiation photographically between ground substance and the other elements.

SUMMARY AND CONCLUSIONS

1. A brief review of the literature on connective tissue ground substance and the spreading factor is presented.

2. The similarity in reaction of the ground substance and the nasal turbinates to various toxic, infectious, or physiological substances is noted.

3. The need for further study of basic nasal physiology is presented, and it is suggested that the newer histochemical stains used in the study of connective tissue would be of value in this work.

4. A series of stains modified to give more satisfactory results with nasal turbinate material are presented.

5. Preliminary study warrants the following conclusions regarding the ground substance of the nasal turbinates: a) The acid mucopolysaccharides consist primarily of the chondroitin sulfate group. b) These are present in a relatively depolymerized form. c) Mast cells are present but scarce.

6. Further work on various functional and pathological states of the nasal turbinates is in progress.

7. It is hoped that the stain techniques and the philosophy of those who have pioneered connective tissue study will be useful in furthering the study of ear, nose, and throat problems.

40 SAN MATEO DRIVE

REFERENCES

1. Duran-Reynals, F.: Exaltation de l'activite du virus vaccinal par les extraits des certain organes. *Compt. rend. soc. biol.* 99:6, 1928.
2. Asboe-Hansen, G.: *Connective Tissue in Health and Disease*. Philosophical Library, N.Y., 1957.
3. Miner, R. W.: The Ground Substance of the Mesenchyme and Hyaluronidase. *Annals N. Y. Acad. of Sci.* 52:Art.7:943-1196, 1950.
4. Ciba Foundation Symposium: *Chemistry and Biology of Mucopolysaccharides*. Little, Brown, and Company, Boston, 1958.
5. Tunbridge, R. E.: *Connective Tissue, A Symposium*. Blackwell Scientific Publications, Oxford, 1957.
6. Duran-Reynals, F.: Tissue Permeability and the Spreading Factors in Infection. *Bact. Rev.* 6:197, 1942.
7. Meyer, Karl: Biological Significance of Hyaluronic Acid and Hyaluronidase. *Physiol. Rev.* 27:335, 1947.
8. Meyer, Karl: The Chemistry of the Ground Substances of Connective Tissue. In *Connective Tissue in Health and Disease*, p. 54, 1957.
9. Persson, Bengt: Studies on Connective Tissue Ground Substance. *Acta Soc. Med. Upsala* 58, Supp. 2:11, 1953.
10. Mayer, R. L., and Kull, F. C.: Influence of Pyribenzamine and Antistine upon Action of Hyaluronidase. *Proc. Soc. Exper. Biol. and Med.* 66:392, 1947.

11. Hechter, O.: Mechanisms of Spreading Factor Action. *Annals N.Y. Acad. Sci.* 52:7:1028, 1950.
12. Dorfman, A.: The Action of Serum on Hyaluronidase. *Ann. N.Y. Acad. Sci.* 52:7:1098, 1950.
13. McMasters, P. D., and Parsons, R. J.: The Movement of Substances and the State of the Fluid in the Intradermal Tissue. *Ann. N.Y. Acad. Sci.* 52:7:992, 1950.
14. Fischel, E. E.: Effect of Salicylate and Tripeleminamine HCL (PBZ) on Arthur Reaction and Bacterial Allergic Reaction. *Proc. Soc. Exper. Biol. and Med.* 66:537, 1947.
15. Sallman, B., and Birkeland, J. M.: The Role of Hyaluronidase in Hemolytic Streptococcal Infection. *Ann. N.Y. Acad. Sci.* 52:7:992, 1950.
16. Pike, R. M.: The Production of Hyaluronic Acid and Hyaluronidase by Some Strains of Group A Streptococci. *Ann. N.Y. Acad. Sci.* 52:7:1070, 1950.
17. Asboe-Hansen, G.: Variability on Hyaluronic Acid Content of Dermal Connective Tissue under the Influence of Thyroid. *Acta Dermat-Vener.* 30:221, 1950.
18. Proud, G. O., and Lange, R. D.: The Effect of Thyroidectomy on the Nasal Mucosa of Experimental Animals. *Laryngoscope* 67:201, 1957.
19. Iversen, K.: Hormonal Influence on Connective Tissue. In *Connective Tissue in Health and Disease*, Phil. Library, N.Y., p. 103, 1957.
20. Rawlins, A. G.: Review of the Corticosteroids—Their Application to Otolaryngology. *Laryngoscope* 66:674, 1956.
21. Lillie, R. D.: *Histopathologic Technique and Practical Histochemistry*. Blakiston Company, Inc., p. 118, 1954.
22. *Ibid.*: p. 355.
23. Pearse, A. G. E.: *Histochemistry, Theoretical and Applied*. J. and A. Churchill, Ltd., London, p. 436, 1954.
24. Lison, L.: Alcian Blue 8G with Chlorantine Fast Red, 5B, *Stain Tech.* 29: 131-138, 1954.
25. Mowry, R. W.: Alcian Blue Techniques for the Histochemical Study of Acidic Carbohydrates. *J. Histochem. and Cytochem.* 4:407, 1956.
26. Kramer, H., and Windrum, G. M.: The Metachromatic Staining Reaction. *J. Histochem. and Cytochem.* 3:227-237, 1955.
27. Rinehart, J. F., and Abul-Haj, S. K.: An Improved Method of Histologic Demonstration of Acid Mucopolysaccharides in Tissues. *A.M.A. Arch. Path.* 52: 189-194, 1951.
28. Gomori, G.: *Microscopic Histochemistry*. Univ. of Chicago Press, Chicago, p. 73, 1952.
29. Pearse, A. G. E.: *Histochemistry, Theoretical and Applied*. J. and A. Churchill, Ltd., London, p. 431, 1954.

XXIV

NONCHROMAFFIN PARAGANGLIOMA OF THE HEAD AND NECK

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In recent years, widespread interest has been reawakened in a group of tumors occurring in the head and neck classified under a variety of names. The terminology we favor is nonchromaffin paraganglioma. Tissues from which these tumors usually arise are contained in the carotid body at the bifurcation of the common carotid artery and in the glomus jugulare at the base of the skull in close relation to the jugular bulb and middle ear. Tumors developing in these areas produce signs and symptoms from involvement of adjacent structures which often are brought to the attention of the otologist.

Benign and malignant neoplasms represent the only pathological lesions of the paraganglia. The relative rarity of these tumors, their classification under a variety of names, their pleomorphic manifestations and clinical evolution, together with the various histological patterns and the possibility of malignant degeneration, make them an especially interesting group of tumors. These many factors have contributed to some confusion regarding diagnosis, prognosis and therapy.

These considerations have stimulated us to review a series of twenty-three patients with histologically verified nonchromaffin paragangliomas of the head and neck and to analyze all of the aural polyps previously diagnosed as benign.

HISTORICAL REVIEW

The carotid body was first mentioned by Von Haller¹ in 1743 as the "ganglion minutum." In 1762 Luschka² gave a more detailed

¹Read before the Sixth International Congress of Otolaryngology, Washington, D.C., May, 1957.

anatomical and microscopic description of the "ganglion intercaroticum" as a part of the sympathetic chain.

The first report of a carotid body and its removal is attributed to Marchand³ in 1891. However, the patient expired on the third postoperative day. Scudder⁴ is believed to have been the first American surgeon successfully to remove the carotid body and verify it histologically. This was in 1903. Since that time a few hundred cases of carotid body tumor have been reported in the medical literature, many with excellent historical recapitulations.

The glomus jugulare in the temporal bone has received extensive recognition as a new entity. However, Valentin⁵ in 1840 described a ganglion-like formation at the initial portion of the tympanic nerve as "gangliolum tympanicum." In 1878 Krause⁶ pictured a formation whose structure was similar to that of the glandula intercarotica and named it "glandula tympanica." The term "paraganglion" was proposed by Kohn⁷ because of its embryological origin from the same anlage as the sympathetic ganglia.

The first accurate description of the "glomus jugulare" was given by Guild^{8,9} in 1941. According to him these structures occur in the adventitia of the dome of the jugular bulb and along the tympanic branch of the glossopharyngeal (Jacobson's nerve) and the auricular branch of the vagus (Arnold's nerve). Other similar structures include the paraganglia intravagale and juxtavagale, the paraganglium ciliare, and the paraganglium aorticum.

All these paraganglia are considered by Lattes¹⁰ as homologous parts of a system of organs consisting of nonchromaffin tissue and closely associated with the parasympathetic division of the autonomic nervous system. They seem to have an embryological development which parallels the branchial vessels and cranial nerves, an analogous histological appearance and a chemoceptive function.

The first case of carotid body-like tumor of the middle ear was reported by Rosenwasser¹¹ in 1944. Since then a widespread interest concerning these tumors has arisen in the world literature. New cases have been reported. Others have been re-evaluated and reclassified.

Several terms have been employed to designate these tumors, chiefly according to their site of origin. Descriptive of this trend are carotid body tumors, glomus jugulare tumors, jugular body tumors and tympanic body tumors. The term chemodectoma was proposed



Fig. 1.—Schüller-Law view of the right mastoid. There is an area of rarefaction of the mastoid tip and cloudiness of the remaining mastoid cells.

by Mulligan¹² because of the assumed chemoreceptor function of the tumor. When we consider that the exact site of origin of the tumor cannot always be ascertained in relation to the normal variations in the location of these structures, and that their function has not yet been definitely established, we prefer to employ the term "nonchromaffin paraganglioma" for all of these tumors because it is more descriptive of the histological characteristics they have in common.

According to some authors,¹³ four types of neoplasms would be derived from the four sections of the functional system associated with the regulation of the circulation of the body: 1) pheocromocytoma from the chromaffin tissue, 2) nonchromaffin paraganglioma, 3) glomangioma from the glomerular arterio-venous anastomoses, 4) pericytoma from the pericytes.

CLINICAL ASPECTS

Many of the signs and symptoms which characterize these tumors are common to benign and malignant neoplasms at the same site.

Others, however, evidence a peculiarity which defies a satisfactory explanation.

Women are more often affected than men in differing numbers according to the various authors. In our series the ratio is approximately 1:5 (19 females and 4 males). The right and left sides appear equally often affected (13 on the right, 9 on the left, and one patient had bilateral involvement in this group). A family history has been noted particularly in the paragangliomas of the temporal bone. The association of multiple nonchromaffin paragangliomas in the temporal bone and the neck has also been observed.¹⁴⁻¹⁶ One of our cases presented a glomus jugulare tumor on the right and a carotid body tumor on the left side.

The life history of the condition, though usually of long duration, is in our opinion strictly related to the histological pattern of the tumor and its evolution.

Nonchromaffin paragangliomas have been described in children of a few months of age and in persons over eighty. The greatest frequency in our group varied from 47 to 77 years of age (Table I).

1) *Nonchromaffin Paragangliomas of the Temporal Bone.* Signs and symptoms of nonchromaffin paragangliomas vary depending on several factors: time at which patients sought medical care, point of

TABLE I
INCIDENCE OF NONCHROMAFFIN PARAGANGLIOMAS
According to Age Group

AGE OF PATIENTS	NUMBER OF CASES
0 - 10	1
11 - 20	0
21 - 30	2
31 - 40	1
41 - 50	3
51 - 60	10
61 - 70	1
71 - 80	5

origin of the tumor, clinical evolution, whether there was expansion in the middle and external ear, or if there was extension to the petrous bone or the base of the skull. Such considerations render a chronological classification impossible.

Symptoms may be classified as otologic, neurologic, and vascular. The presenting complaints of our cases are reported in Table II. The

TABLE II
PRESENTING SYMPTOMS
(In 18 cases of Nonchromaffin Paragangliomas
of the Temporal Bone)

	CASES	PER CENT
Hearing Loss	16	88
Otorrhea	11	61
Earache	9	50
Tinnitus	7	38
Vertigo	4	22
Facial Paralysis	4	22
Headache	4	22
Growth in the Ear	2	11
Postauricular Swelling	1	5
Twitching of Face	1	5
Exophthalmos	1	5

symptoms commonly reported are hearing loss, otorrhea, otalgia, tinnitus and headache.

The progressive loss of hearing which is unilateral is present in 88 per cent of the patients analyzed. Otorrhea represents approximately 61 per cent, frequently associated with a hemorrhagic component (otorrhagia), especially following manipulation. One-half of the patients complained of earache or postauricular pain, most likely due to pressure by the tumor or secondary infection. Tinnitus (38 per cent) was described by the patients as a pounding, pulsating or blowing noise on the ipsilateral side and usually of rather long duration. Dizziness was variously described as a true vertigo in those instances

in which the petrous portion of the temporal bone was involved. Headache in four cases was referred to the vertex, occurred as a dull persistent ache, probably a result of pressure.

The otological examination (Table III) revealed injection of the lower quadrants of the tympanic membrane or a bulging, bluish dis-

TABLE III
OTOLOGIC FINDINGS
(In 18 cases of Nonchromaffin Parangliomas
of the Temporal Bone)

	CASES	PER CENT
A. OBJECTIVE		
Polypoid Mass in External Auditory Canal	14	78
Tenderness over Mastoid	3	16
Discolored Tympanic Membrane	2	11
Bulging Tympanic Membrane	2	11
Postauricular Swelling	2	11
Bruit over Mastoid	2	11
B. FUNCTIONAL		
Hearing Loss	17	94
Severe	14	78
Complete	3	16
Absence of Vestibular Response	3	16

colored drum in four cases. A myringotomy at this stage may be followed by profuse bleeding. This was the experience of several of our patients who apparently were thought to have acute otitis media. At a later stage, a polypoid, richly vascular, easily bleeding mass (14 cases) may appear in the external auditory canal, with the pulsation being noted on the slightest compression with the Brünig otoscope. The mass may evidence a slow, but progressive expansion. Postauricular swelling was present in two cases associated with subperiosteal abscess.

Functional tests, cochlear and vestibular, revealed marked hearing loss (14 cases) of a conductive or of a mixed type (presbycusis) but

with predominance of the conductive component. The vestibular response was normal. In three other cases complete unilateral hearing loss was associated with absence of vestibular reaction, neurological signs, and radiologic and operative evidence of petrous bone involvement.

Neurological symptoms reflect paresis or paralysis of the facial and hypoglossal nerves (Table IV). The facial weakness or palsy is

TABLE IV

NEUROLOGIC FINDINGS

(In 18 cases of Nonchromaffin Paragangliomas
of the Temporal Bone)

	CASES	PER CENT
Total Number of Cases with Neurologic Findings	6	33
Facial Paralysis	4	22
Hypoglossus Involvement	3	16
Facial Paresis	2	16
Foramen Lacerum Posterius Syndrome (IX, X, XI)	1	5
Trigeminal Involvement	1	5
Exophthalmos	1	5

of the peripheral type. Twitching of the facial muscles was observed in one case. Less frequently, involvement of the glossopharyngeus, vagus and spinal accessory nerves has been noted as part of a jugular foramen syndrome. Involvement of the III and VI cranial nerves as well as Horner's syndrome and trigeminal neuralgias has also been reported.¹⁷ Cranial nerves are usually affected on the same side as the lesion. Such paralyzes develop gradually and progress slowly.

As the neoplasm expands, the pontine-cerebellar angle and the posterior cranial fossa may be invaded. Occasionally, intracranial hypertension may develop and the tumor may simulate a brain abscess or brain tumor.¹⁸ This was observed in one of our cases and confirmed at autopsy as an edematous brain, however without invasion by the neoplasm which had eroded the superior surface of the petrous bone.



Fig. 2.—Schüller-Law view revealing a large destructive process over the mastoid and involving the tegmen and the sinus wall.

The vascular symptoms include turgescence of the superficial cervical veins, particularly of the external jugular vein, or in some patients a bruit may be heard over the mastoid region. The bruit may decrease in intensity or disappear with pressure over the common carotid artery on the same side.

2) *Nonchromaffin Paragangliomas of the Cervical Region.* The symptoms of paragangliomas of the cervical region are produced chiefly by expansion of the neoplasm and the relationship of the lesion with the pharyngeal wall and the neurovascular bundle of the neck. The tumor is usually painless and asymptomatic, but at times there is a vague discomfort in the distribution of the cervical plexus or a pain referred to the ear. Because of pressure on nearby structures, there may be hoarseness, cough, dysphonia, dysphagia, nausea, vomiting, headache and tinnitus. A hyperactive carotid sinus syndrome may be demonstrated. One of us (FLL) has observed a patient with bilateral involvement.

On physical examination a tumor mass may be palpable in the upper, anterior, cervical triangle under the sternocleidomastoid muscle.



Fig. 3.—Towne's view showing an extensive destruction of the mastoid process with involvement of the tegmen. There is also a marked enlargement of the internal auditory canal and involvement of the labyrinth.

A mass of variable size is usually found which is round, firm, smooth, non-adherent to the skin and movable laterally but not vertically. The tumor may transmit pulsation, but it is not expansile. Such pulsation disappears or decreases with compression of the common carotid artery.

The neoplasm may protrude as a bulging mass on the lateral wall of the oro- or nasopharynx. There may be involvement of the vagus, glossopharyngeus and sympathetic nerves with an associated Horner's syndrome.

Differential diagnosis should include: 1) aneurysms, which are soft and evidence expansile pulsation; 2) primary or metastatic malignancies, which usually are stony hard, irregular and nodular; 3) lymphomas, which may show involvement of other groups of nodes and changes in blood morphology; 4) branchial cysts, which are soft and cystic but rarely bulge into the pharynx; 5) acute and chronic lymphadenitis, with specific and nonspecific inflammatory signs; and

6) neurofibromas, which are laterally placed and are clinically difficult to differentiate.

Exploratory needle biopsy has been described, but may be associated with danger in the region of the great vessels and nerves of the neck.

RADIOLOGIC ASPECTS

Radiological studies of the skull and temporal bone are important aids both in the diagnosis and in the follow up of these tumors, especially those occurring in the middle ear. Routine views should be taken to be supplemented by special projections as indicated.

The essential findings of changes in our cases may be classified as follows (Table V):

TABLE V

RADIOLOGIC FINDINGS

(In 18 cases of Nonchromaffin Paragangliomas
of the Temporal Bone)

MASTOID PROCESS (15 cases—83%)

- Sclerosis
- Cloudiness
- Cellular Destruction
- Destruction of Tegmen
- Destruction of Sinus Plate

PETROUS BONE (3 cases—16%)

- Destruction of Semicircular Canals
- Destruction of Cochlea
- Destruction of Apex
- Enlargement of the Internal Auditory Meatus

JUGULAR FORAMEN (2 cases—11%)

- Enlargement with Erosion of Adjacent Bones

a) In the mastoid process. Early, only mastoid sclerosis or clouding is to be observed (Fig. 1). As a result the condition is often



Fig. 4.—Petrous, axial vertico-submental view. There is a prominent enlargement of the right jugular foramen with erosion of the adjacent bones and of the apex of the petrous bone.

diagnosed as chronic mastoiditis. The mastoid process may also appear normal. Later, cellular destruction of the mastoid becomes apparent (Fig. 2).

b) In the petrous bone. With the growth of the neoplasm the petrous ridge may be destroyed and the tumor invade the middle fossa. In other cases the apex of the petrous bone appears to be destroyed. A widening of the internal auditory meatus has been observed. In such instances the radiologic appearance may be that of an acoustic neuroma (Fig. 3).

c) At the jugular foramen. Full axial views are necessary to visualize clearly the basilar foramina. The jugular foramen may appear enlarged when compared to the opposite side or there may be expansion with erosion of the adjacent bones (Fig. 4).

Angiographic studies have been performed through the common carotid and the vertebral arteries, but may be significant only in the

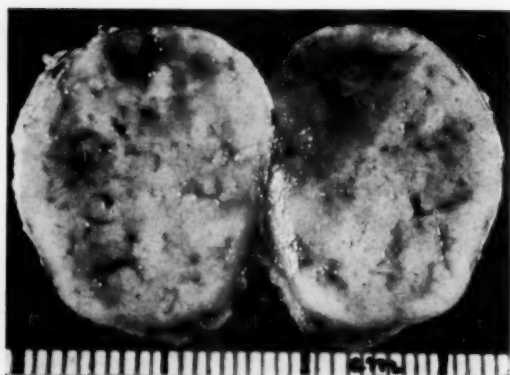


Fig. 5.—Nonchromaffin paraganglioma of the neck. The ovoid mass is surrounded by a fibrous capsule. The cut surface is homogeneous and small blood vessels as well as areas of hemorrhagic necrosis are clearly visible.

presence of an extension into the cranial fossae. Angiograms are most important in differentiating nonchromaffin paragangliomas from acoustic neurinomas. A neurinoma usually does not receive such an abundant vascularization from branches of the external carotid artery. Hooper¹⁹ has studied angiograms through the external carotid artery and outlined the characteristic changes as follows:

- a) Increase in number and size of branches of the external carotid artery to the temporal bone region.
- b) Increased density of the temporal bone due to the presence of the contrast material.
- c) Small vascular channels and loops around the fringe of the shadow of the temporal bone.
- d) Presence of arteriovenous anastomoses.
- e) Evidence of obstruction of the lateral or sigmoid sinus and reversal of flow in this channel.

The radiological studies of nonchromaffin paragangliomas of the neck are usually not diagnostic. Due to the possibility of expansion of the neoplasms in the soft tissues of the neck and the lateral wall of the pharynx, invasion of bony structures is rare. Encroachment of the tumor mass on the naso- and oropharyngeal airway may be demon-

strated. If the tumor invades the base of the skull, bony destruction is evident.

PATHOLOGIC ASPECTS

The macroscopic appearance of nonchromaffin paraganglioma of the neck is that of a globular or ovoid reddish-gray mass of homogeneous consistency, varying from rubbery to soft. The relationship of the tumor to the large vessels in the neck is most often so intimate that the neoplasm cannot be easily dissected. In other instances, the tumor dissected from the carotid arteries shows a groove due to pressure by the vessels. A fibrous capsule surrounds the tumor; thin-walled blood vessels may be observed on the surface. The cut surface is homogeneous, greyish-red, and small blood vessels, as well as areas of hemorrhagic necrosis, may be observed (Fig. 5).

Nonchromaffin paragangliomas of the temporal bone appear macroscopically as aural polyps because their expansion is influenced by the bony structures which surround them. The tumor originates in the middle ear and extends along paths least resistant: outward into the external auditory canal and inward through the eustachian tube and mastoid process. The tumor may also involve the foramen lacerum posterius and extend into the neck, simulating a cervical tumor.

When the petrous bone is invaded by the neoplasm there is usually evidence of progressive bony destruction, characterized by the lack of defensive reaction and new bone formation. The labyrinthine spaces become filled with blood or tumor tissue. However, just the very opposite process, namely, that of ossification of the labyrinth, probably as an expression of labyrinthitis ossificans, has also been reported.²⁰

Several histological classifications of nonchromaffin paragangliomas have been proposed according to their cellular arrangement and abundance of blood vessels. Three forms have been considered:²¹ a) normal type, reproducing the paraganglion structures; b) adenomatous type, with the formation of glandular pseudoacini; and c) angiomatous type, with predominance of the vascular component.

A few basic principles need to be considered in the histological diagnosis of these tumors: 1) the characteristics of the cellular elements and the vascular stroma are fundamental for the diagnosis; 2) the several histological patterns, organoid and angiomatous, may be associated, giving the tumor a pleomorphic appearance; 3) the frequent hemorrhagic phenomena within the tumor, associated with

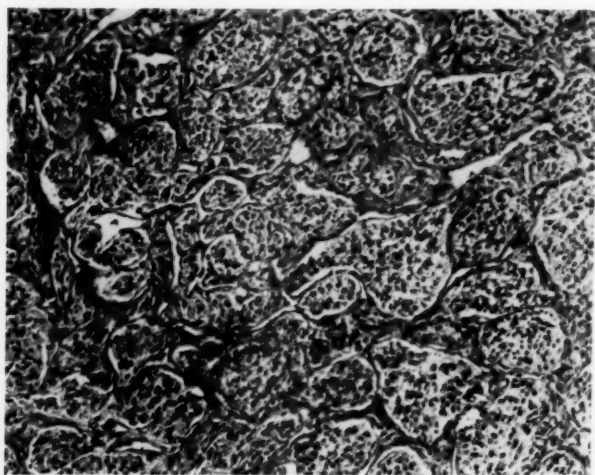


Fig. 6.—Photomicrograph, nonchromaffin paraganglioma of the neck showing the typical arrangement of nests of epithelioid cells surrounded by capillary sinuses.

connective tissue reaction, may alter the typical histological picture of these growths and 4) phenomena of degeneration, secondary suppurative inflammation and the presence of bizarre, multinucleated giant cells, may further complicate the microscopic pattern.

The cellular elements are fairly uniform in size, and round or polyhedral in shape. As in the normal paraganglia, two types of epithelioid cells are observed in these tumors: one with a large vesicular nucleus limited by a well-defined nuclear membrane and containing the nuclear chromatin in small clumps and one or more nucleoli; the other with small hyperchromatic pyknotic nuclei and more abundant cytoplasm. The cytoplasm stains lightly with eosin and is occasionally faintly granular. Mitotic figures are rarely observed.

The vascular stroma is variable, being scanty in some areas and very rich in others. The vascular channels are usually lined with a single layer of endothelial cells which may be deficient. Silver staining reveals a fine lacy argentaffin fibrillar network surrounding the nests of cells and vascular channels.

The epithelioid cells may accumulate in islands of various size and shape (organoid pattern) separated from one another by the vascular network; in other areas the cells collect in a strand-like formation (peritheliomatous pattern). In some cases thick bands of fibrous connective tissue irradiate from the capsule and separate the islands of cells (pseudoacini). This morphologic appearance has been interpreted as a connective tissue reaction to a hemorrhagic phenomenon as demonstrated by the presence of old blood pigment in this fibrous stroma. Areas of hyaline degeneration are displayed by some of the tumors. In others irregular, multinucleated giant cells are observed. These have been considered as a characteristic of malignancy (Figs. 6 - 10).

The microscopic picture of nonchromaffin paraganglioma has apparently, at least in the past, resulted in such diagnoses as angioma, angiosarcoma, angiofibroma, endothelioma, and hemangioendothelioma. They were evolved mainly by considering the tumor to be of vascular origin. The angiomatous type of nonchromaffin paraganglioma may be differentiated from simple angiomas by the abundance of cellular elements of characteristic morphology among the rich vascular network. In comparison with angiosarcomas, nonchromaffin paragangliomas demonstrate a greater uniformity of cells, rare atypical forms and mitoses. Differentiation from hemangioendotheliomas is sometimes difficult. The morphologic appearance of the cells and the argentaffin fibrillary network which surrounds the cell nests along the vascular channels, are often useful criteria.²²

Because of the tendency to hemorrhage, progressive expansive growths with bone destruction and involvement of vital structures, and the possibility of metastases, nonchromaffin paragangliomas should be considered as histologically benign, but clinically malignant tumors of the head and neck.

The possibility of a correlation between the histological pattern and the clinical behavior in these tumors has been suggested. The presence of irregular, multinucleated giant cells has been considered as evidence by some investigators of malignancy. Other authors²³ have denied any such correlation. It has been our observation that a more fibrous or acellular structure is less likely to have an invasive character, while nonchromaffin paraganglioma of a more cellular type with many vascular channels coursing irregularly through them have demonstrated a more rapid tendency to expansion and invasion of adjacent structures.

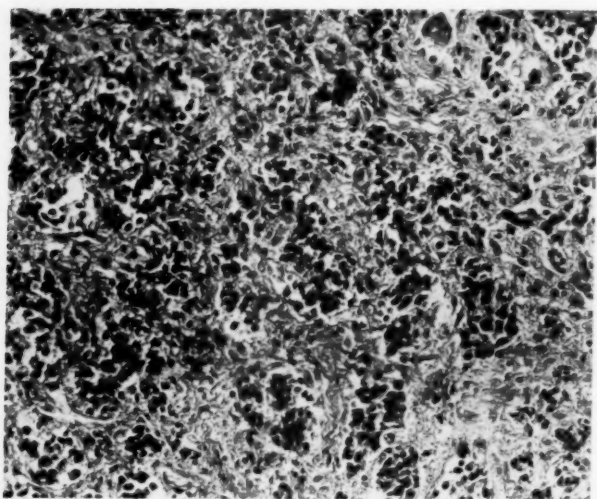


Fig. 7.—Nonchromaffin paraganglioma of the temporal bone. The islands of epithelioid cells are surrounded by thick bands of fibrous connective tissue (pseudoacini).

THERAPEUTIC ASPECTS

The therapeutic approach, both surgical and radiological, should be evaluated according to the following selective factors: a) end result in a long term follow up, palliation with symptomatic relief; b) morbidity and mortality of the procedure; c) functional and cosmetic results; d) physical and emotional strain; and e) availability of equipment and trained personnel.

1) *Nonchromaffin Paragangliomas of the Temporal Bone.* Evaluation of therapeutic results of these tumors is usually very difficult. Their rate of growth is very slow and the clinical course and appearance may be stationary over a long period of time. As a corollary, the end-result in fixing a five-year period is not satisfactory to judge a complete cure. Graf,²⁴ in reviewing the literature, has shown that glomus jugulare tumors have a good short term, but a definitely poor long term prognosis. It usually takes 15 to 20 years or longer before the infiltrative tumor leads to death. The management of these lesions should consider an adequate preoperative workup, the surgical approach to an isolated or extensive tumor, the palliative measures, the

dangers and safeguards of the procedures, and the end results. Of primary importance is the preparation of the patient for massive blood transfusions in all procedures.

The problem of the seriousness of the preoperative biopsy of these tumors is to be considered most carefully because of the possibility of massive hemorrhage. Suppuration and granulation tissue may disguise the appearance and true nature of the neoplasm. Adequate biopsy should be performed in the operating room by snare or punch forceps technique. Inadequate biopsy often reveals only a red friable tissue, usually diagnosed as chronically infected granulation tissue. However, the diagnosis should be entertained on the basis of the clinical examination and corroborated by biopsy.

The treatment of choice of these tumors of the temporal bone is wide surgical exposure with complete radical resection. Surgery is a necessity in patients with associated suppuration. However, the extent of the lesion should be determined as well as possible preoperatively by clinical and radiological interpretation.

If the tumor is small and of limited extent, surgical excision is practical without undue risk or difficulty. Though very early glomus jugulare tumors may be removed by hypotympanotomy,²⁵ a radical mastoidectomy through an endaural or postauricular approach is most often mandatory. Complete removal and eradication of the tumor is more important than preservation of function in these patients. In view of its marked hemorrhagic tendency, the tumor should be exposed from all sides if possible, before removal is attempted.

When the neoplasm invades the petrous bone or destroys the tegmen or the sinus plate, a labyrinthectomy and ligation of the external carotid artery and internal jugular vein may be necessary. Because of the overabundant blood supply of these tumors by way of the external carotid artery, ligation of this vessel is thought to be of value in slowing down the rate of growth.

If the petrous bone is extensively involved and there is no possibility of successful removal, palliative x-ray therapy should be the treatment of choice. In instances of incomplete removal, postoperative irradiation and radon seeding is indicated. It is generally agreed that radio- and roentgentherapy may decrease the size of the tumor, but are ineffective in preventing remission. Williams et al²⁶ have recently reported definite favorable effects following radiation therapy.

TABLE VI
EVALUATION OF THERAPY OF NONCHROMAFFIN PARAGANGLIOMA OF THE TEMPORAL BONE
Part I: *Surgery*

CASE	AGE	PROCEDURE	FINDINGS	COURSE	FOLLOW UP
1.	52	Rad. Mastoid.	Mass in antrum & c. tympani	Temp. VII p.; no recurrence	31 years
2.	33	Polypectomy & hypotympanotomy	Mass in ext. can.	No recurrence	9 years
3.	72	Rad. Mastoid.	Mass in antrum & c. tympani	Electrocoagulation of persistence; VII paralysis; no recurrence	8 years
4.	55	Rad. Mastoid.	Mass in attic & c. tympani	No recurrence	6 years
5.	67	Rad. Mastoid.	Mass in mastoid & c. tympani	No recurrence	5 years
6.	48	Rad. Mastoid.	Mass in cavum tympani	No recurrence	2 years
7.	50	Rad. Mastoid.	Mass in cavum tympani	No recurrence	2 years
8.	59	Rad. Mastoid.	Mass in antrum & c. tympani	No recurrence	2 months
9.	73	Rad. Mastoid. Labyrinthectomy	Destruction of mastoid, tegmen, sinus plate, fac. canal & labyrinth with suppuration	Died (meningitis) 9 days postop. Autopsy: metastases to liver and lungs	Dead

TABLE VII
EVALUATION OF THERAPY OF NONCHROMAFFIN PARAGANGLIOMA OF THE TEMPORAL BONE
Part II: *Surgery and Irradiation*

CASE	AGE	PROCEDURE	FINDINGS	IRRADIATION	COURSE	FOLLOW UP
1.	26	Rad. mastoid	Mass in mastoid & chorda tympani	Radon s. 270 mc/hrs. x-ray: 610 r	Recurrence 5 years later Rev. of mastoid; Perm. VII p. No recurrence	21 years
2.	71	Rad. mastoid	Mass in mastoid & chorda tympani	4000 r preop. 3000 r postop.	No recurrence	10 years
3.	56	Rad. mastoid mastoid &	Destruction of mastoid & fac. can. suppur.	1500 r	Serous labyrinth. postop. No recurrence	7 years
4.	57	Rad. mastoid	Mass in antrum & c. tympani. Facial exposed	3000 r postop.	VII paralysis after x-ray No recurrence	4 years
5.	56	Rad. mastoid	Mass in mastoid & c. tympani. Fac. can. destroyed	6000 r postop.	No recurrence	4 years
6.	49	Excision of lesion at jug. foramen— Rad. mastoid	Cervical mass thru jug. foramen. Mass in c. tymp. destruction of petrous apex	2000 r postop.	X paralysis postop. Died 2 years later Autopsy: intracranial extension No distant metastases	2 years Dead
7.	58	Rad. mastoid	Mass in antrum & c. tympani	5200 r postop.	No recurrence	6 months

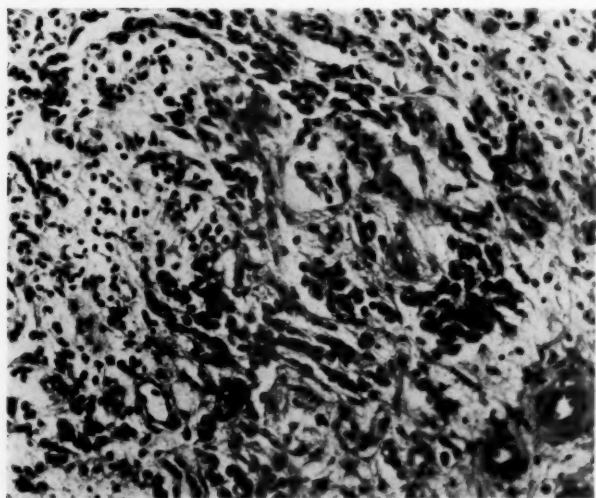


Fig. 8.—Nonchromaffin paraganglioma of the temporal bone. Peritheliomatous aspect of neoplasm with a few island formations.

It is our contention, however, that the combination of wide surgical resection followed by postoperative irradiation yields the best results in extensive lesions.

An evaluation of the end result of our cases of nonchromaffin paraganglioma of the temporal bone is reported in Tables VI and VII. Two of the patients refused surgery after aural polypectomy and show evidence of progressive expansion. The remaining sixteen cases were divided into two groups: one group of patients who were treated by surgery alone (nine cases), and the other by surgery and irradiation (seven cases). The follow-up varied from 31 years to two months.

The patients treated by surgery alone had the benefit of a radical mastoidectomy with the exception of one patient who had an endaural polypectomy and hypotympanotomy. A clinical and radiological evaluation at different intervals failed to reveal any evidence of recurrence in seven of the patients. One of the remaining two patients showed persistent tumor which was electro-coagulated and is now free of disease. The other patient had a radical mastoidectomy and labyrinthectomy because of extensive destruction of the mastoid and

petrous bone. This patient succumbed to meningitis nine days postoperatively. The autopsy revealed metastases to the liver and the lungs.

The patients treated with surgery and irradiation had more extensive lesions than the preceding group. All of them had a radical mastoidectomy. One patient first presented a cervical mass which protruded through the jugular foramen and was excised through a cervical approach. Irradiation included both x-ray therapy and radon seeding; dosage varied from 1500 r to 7000 r.

TABLE VIII
COMPLICATIONS

SURGERY	
Facial Paralysis	
Temporary	1
Permanent	2
Serous Labyrinthitis	1
Vagus Nerve Involvement	1
IRRADIATION	
Facial Paralysis	1

The irradiation was usually given postoperatively, except in one case which had x-ray therapy pre- and postoperatively. Five of these patients showed no evidence of disease at varying intervals. One patient had persistence five years after surgery and irradiation; a revision of the mastoidectomy was performed and after 16 years there is absence of tumor. The patient with a cervical extension through the jugular foramen had persistent tumor at all times; she died two years later with signs of increased intracranial pressure. The autopsy revealed an intracranial extension of the tumor through the middle fossa, but no sign of brain invasion and no distant metastases.

The choice between surgery and surgery plus irradiation should be made individually after careful evaluation of the extent of the lesion. If possible, surgery should be performed first, followed by irradiation of the persistent tumor. Both surgery and irradiation present hazards that should be evaluated in advance (Table VIII). In our group, one patient developed a temporary serous labyrinthitis and another a temporary facial paralysis following radical mastoidectomy.

Two other patients had a permanent facial paralysis following a wide revision of the mastoidectomy and electrocoagulation of the persistent tumor. A paralysis of the X cranial nerve followed the cervical procedure for excision of the extension through the jugular foramen. Only one patient developed a permanent facial paralysis following deep x-ray therapy.

A careful, long term follow-up, with biopsy of all suspicious tissue, is mandatory.

In patients judged to be terminal, the control of pain can be achieved to a degree by ligation of the external carotid artery.

2) *Nonchromaffin Paragangliomas of the Neck.* The ideal treatment of these tumors is excision without injury to any of the neurovascular structures of the neck. As a matter of fact, the hazardous factor in the surgical treatment of these neoplasms lies in the ligation of the common or the internal carotid artery. Carotid compression tests and angiographic studies have been proposed to reveal advanced evidence of the presence of abnormalities, irritable carotid sinus, cerebrovascular insufficiency from various causes or a combination of both. However, no method can be considered so reliable at this time so as to predict which patient will or will not tolerate ligation. The possibility of thrombosis is still a potential threat. In one of our patients the internal carotid artery appeared very stenotic and loose. Its ligation was not followed by any untoward effect.

As a general rule any patient in whom a nonchromaffin paraganglioma of the neck is suspected, exploration is in order because the clinical diagnosis is, in most instances, inaccurate. The psychological preparation of the patient for the seriousness of the condition and the typing and cross matching of blood are of utmost importance.

When the tumor has been identified and clearly exposed it should first be separated from all surrounding soft tissues and then dissected away without injury to the internal or common carotid arteries if at all possible.

If the tumor is firmly adherent to the carotid arteries, the dissection should be carried out meticulously in the plane of arterial adventitia in a bloodless field. Continuous aspiration is a necessity. Embryological studies have demonstrated that the carotid body arises in the adventitia of the carotid artery. Such relationship is maintained by nonchromaffin paragangliomas. It has also been demon-

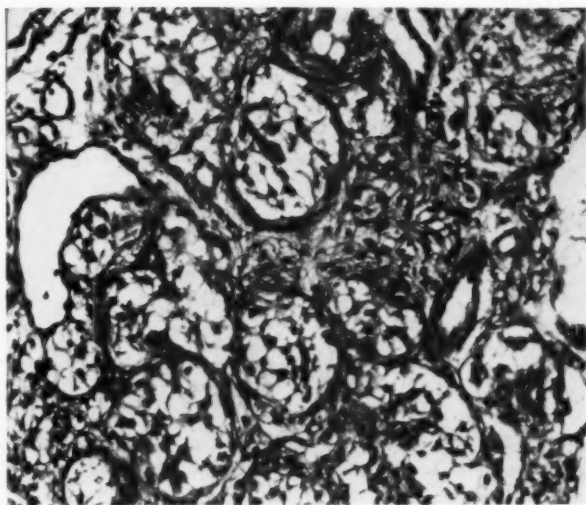


Fig. 9.—Nonchromaffin paraganglioma of the temporal bone showing the two types of epithelioid cells observed in these tumors. The vascular channels are lined with a single layer of endothelial cells.

strated²⁷ that the adventitia of large and medium sized arteries can be sacrificed without endangering the result of primary anastomosis or arterial homografts. The eventual restoration of continuity of arterial walls, therefore, should be carried out by simple closure of the defect, primary, end-to-end anastomosis, or by the insertion of an arterial homograft or venous autograft.

Though a certain percentage of nonchromaffin paragangliomas may undergo malignant transformation or may evidence local invasion, the ligation of the common carotid or internal carotid entails too high a postoperative mortality and danger of cerebral damage to be justified. If the tumor cannot be resected, a biopsy should be obtained and careful hemostasis supplied. In most of these patients, as well as in cases of incomplete removal, palliative therapy should be employed using radon seeding, x-ray, betatron, or cobalt therapy. By means of such modalities, the growth of the tumor can be reasonably slowed down to avoid distressing symptoms, the patients presenting only a cosmetic defect, dysphonia and dysphagia.

The problem arises as to whether the ligation of the common or internal carotid should be justified if the tumor presents local invasive character and the histological appearance is bizarre and highly cellular to suggest malignancy. Though no definite correlation has yet been established between the histological appearance and prognosis, it is our opinion that such patients should be evaluated individually and that the rationale for intervention should be modified in accordance with the exceptional nature of these tumors.

CONCLUSIONS

Increasing interest in the world literature of tumors originating from nonchromaffin paraganglia of the head and neck prompted a review of a series of twenty-three cases observed at the Illinois Eye and Ear Infirmary and the Research and Educational Hospitals of the University of Illinois.

Several terms have been employed in the past to designate these tumors, some according to their site of origin and others which are more descriptive of their histological characteristics. Carotid-body like tumor and glomus jugulare tumor are in most common usage. Since these neoplasms arise from the nonchromaffin paraganglia they should be so designated, nonchromaffin paraganglioma.

The histogenesis of these tumors is analyzed in the light of previous investigations of the normal embryology and morphology of nonchromaffin structures. Nonchromaffin paraganglia are small organs distributed along the great vessels of the neck and some cranial nerves. These structures have no affinity for chromium salts and possess no hormonal secretion, but do retain a chemoceptor function. The most important nonchromaffin paraganglia in the head and neck areas are the carotid, jugular, tympanic, intravagal and juxtavagal, aortic, and ciliary.

The most characteristic clinical and pathological aspects are presented, comparing previous reports with our personal experiences. Clinical symptomatology varies with the site of origin and the evolution. Tumors originating from the tympanic and jugular nonchromaffin paraganglia may simply expand in the middle and external ears or involve the petrous bone and base of the skull. Symptoms may be divided into otological, neurological and vascular.

The symptoms vary in duration from months to many years. The tympanic membrane may appear discolored (bluish) and even bulging.

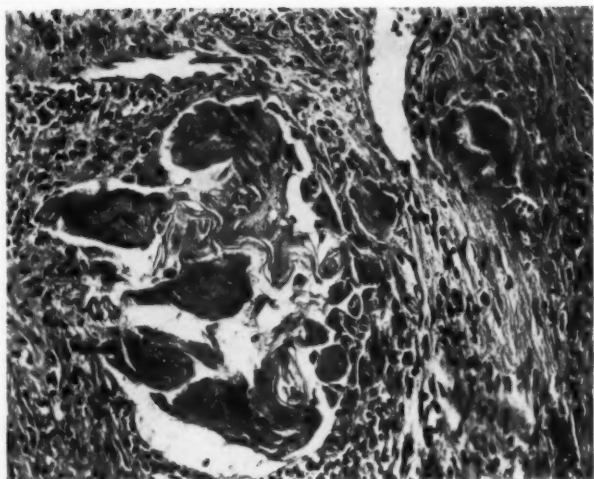


Fig. 10.—Nonchromaffin paraganglioma of the temporal bone. Irregular, multinucleated giant cells are observed in a strand-like formation of epithelioid cells.

A polypoid mass, grayish to red in color, soft or firm, which pulsates on compression, may occupy the external canal. Nonchromaffin paragangliomas of the neck are usually represented by a tender, firm mass associated with bulging of the pharyngeal wall and carotid sinus syndrome.

The radiological examination is not characteristic; however, early there is slight cloudiness, and later extensive destruction. Pathologically, nonchromaffin paragangliomas are benign tumors that may be represented by several types: a) normal, b) adenomatous, and c) angiomatous. The frequent hemorrhagic phenomena within the tumor associated with connective tissue reaction are likely to alter the typical histologic appearance of these tumors.

Microscopically, two kinds of epithelioid cells are seen in these tumors. One has an abundant cytoplasm and a pyknotic nucleus; the other contains scant eosinophilic cytoplasm and a vesicular nucleus. Mitotic figures are rarely observed. The vascular network is very rich and with a thin endothelial lining. Because of the bleeding tendency, progressive expansive growth with bone destruction and involvement

of vital structures, and the possibility of metastases, nonchromaffin paragangliomas are to be considered histologically benign, but clinically malignant tumors of the head and neck.

The therapeutic approach, both surgical and radiological, is evaluated according to the following selective factors: end result in a five-year period, palliation with symptomatic relief, morbidity and mortality of the procedure, functional and cosmetic results, physical and emotional strain, availability of equipment and trained personnel. When possible, thorough surgical exeresis is the treatment of choice. Radiosensitivity of these tumors varies with the histological pattern. Following incomplete removal, roentgen and radium therapy may be employed. Ligation of the external carotid artery is resorted to as a palliative measure for the control of pain.

Nonchromaffin paraganglioma of the head and neck is a clinico-pathological entity in the domain of otolaryngology, but its differential diagnosis and treatment require a comprehensive knowledge of its clinical and microscopic manifestations, and a prolonged follow-up of these patients.

1853 WEST POLK ST.

REFERENCES

1. VonHaller, Albrecht: De vera nervi intercostalis origine. Gottingen, 1743. De nervorum in arterias imperio. Gottingen, 1744.
2. Luschka, H.: Ueber die drüsenartige Natur des sogenannten Ganglion Inter-caroticum. Arch. f. Anat. Physiol. u. Wissenschaftl. Med. 4:405-414, 1862.
3. Marchand, F.: Beiträge zur Kenntnis der normalen und pathologischen Anatomie der Glandula Carotica u. die Nebennieren. Internat. Beitr. z. Wissensch. Med. Festschr. Rudolph Virchow, 1:535, 1891.
4. Scudder, L. C.: Tumor of the Intercarotid Body: A Report of One Case Together with All Cases in the Literature. Am. J. M. Sc. 6:384-389, 1903.
5. Valentin, G.: Ueber eine gangliöse Answellung in der Jacobsonschen Anastomose des Menschen. Arch. f. Anat. Physiol. u. Wissensch. Med. 287-290, 1840.
6. Krause, W.: Handbuch der Menschlichen Anatomie, 3rd Aufl. Hahnsche Buchhandlung, pp. 860-861, Hanover, 1879.
7. Kohn, A.: Die Paraganglien. Arch. f. mikr. Anat. 62:263-265, 1903.
8. Guild, S. R.: A Hitherto Unrecognized Structure, the Glomus Jugularis in Man. Anat. Rec. Supp. 2:28, 1941.
9. Guild, S. R.: Glomus Jugulare, a Nonchromaffin Paraganglion, in Man. ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY 62:1045-1071, 1953.
10. Lattes, R.: Nonchromaffin Paraganglioma of Ganglion Nodosum, Carotid Body and Aortic-arch Bodies. Cancer 3:667-694, 1950.

11. Rosenwasser, H.: Carotid Body Tumor of the Middle Ear and Mastoid. *Arch. Otolaryng.* 41:64-67, 1945.
12. Mulligan, R. M.: Chemodectoma in the Dog. (Abstract) *Am. J. Path.* 26: 680, 1950.
13. Sirtori, C., and Veronesi, U.: Considerazioni morfologiche ed istogenetiche sui tumori glomici in base ad otto osservazioni personali. *Tumori* 60:583-608, 1954.
14. Kipkie, G. F.: Simultaneous Chromaffin Tumors of the Carotid Body and the Glomus Jugularis. *Arch. Path.* 44:113-118, 1947.
15. Bickerstaff, E. R., and Howell, J. S.: The Neurological Importance of Tumors of the Glomus Jugulare. *Brain* 76(part 4):576-593, 1953.
16. Conley, J. J.: Multiple Paragangliomas in the Head and Neck: Case Report. *ANNALS OF OTOLGY, RHINOLOGY AND LARYNGOLOGY* 65(2):356-360, 1956.
17. Siekert, R. G.: Neurologic Manifestations of Tumors of the Glomus Jugulare, Chemodectoma, Nonchromaffin Paraganglioma or Carotid Body-like Tumor. *A.M.A. Arch. Neur. Psych.* 76(1):1-13, 1956.
18. Dockerty, M. B., Love, G. J., and Patton, M. M.: Nonchromaffin Paraganglioma of the Middle Ear; Report of a Case in Which the Clinical Aspects Were Those of a Brain Tumor. *Proc. Mayo Clinic* 26:25-32, 1951.
19. Hooper, R. S.: The Glomus Jugulare Tumor; Clinical and Radiological Features. *J. Fac. Radiologists* 7(2):77-89, 1955.
20. Bast, T. H.: Ossification of the Labyrinth in a Case of Glomus Jugulare Tumor. Paper presented at the Chicago Laryngological and Otolological Society (Feb.) 1957.
21. LeCompte, P. M.: Tumors of the Carotid Body. *A. J. Path.* 24:305-321, 1948.
22. Rossi, G.: I paragangli non cromaffini e la loro patologia neoplastica. *Minerva Otorinolaringologica* 4:1-60, 1954.
23. Pettet, J. R., Woolner, L. B., and Judd, E. S.: Carotid Body Tumors (Chemodectomas). *Ann. Surgery* 137:465-477, 1953.
24. Graf, K.: Therapie und Prognose der Nichtchromaffinen Paragangliome des Ohres. *Practica Oto-rhino-laryng.* 15:284-293, 1953.
25. Shambaugh, G. E.: Surgical Approach for So-called Glomus Jugulare Tumors of the Middle Ear. *Laryngoscope* 65:185-198, 1955.
26. Williams, H. L., Childs, D. S., Jr., Parkhill, E. M., and Pugh, D. G.: Chemodectomas of the Glomus Jugulare (Nonchromaffin Paragangliomas) with Especial Reference to Their Response to Roentgentherapy. *ANNALS OF OTOLGY, RHINOLOGY AND LARYNGOLOGY* 64:546-566, 1955.
27. Morphit, M. H., Swan, H., and Taylor, E. R.: Carotid Body Tumors: Report of 12 Cases, Including One Case with Proved Visceral Dissemination. *A.M.A. Arch. Surgery* 67:194-214, 1953.

THE CLINICAL MASKING LEVEL

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The indications for use of masking and the levels employed in everyday practice vary considerably. The purpose of this paper is to recommend the routine use of 100 db re .0002 dynes/cm² of white (random) noise in both air and bone conduction measurements. This level is selected because it is the maximum intensity that the average patient will tolerate and approaches the hazardous level. According to Air Force regulations an exposure to sound intensities of 107 db re .0002 dynes/cm² per octave band should be limited to 30 minutes.⁴ It is also necessary to recognize the limitations of this form of masking and to seek more effective methods.

The effectiveness of masking, i.e., the ability to eliminate a stimulus is not proportional to loudness. Masking efficiency is the relation of masking ability to loudness or intensity.⁵ The most efficient masking sound is one that produces a large masking effect with minimum intensity. The most efficient masking sound is one with the same frequency characteristics as the stimulus. In the Stenger test for example only a few db difference in loudness between a stimulus presented simultaneously to both ears will cause the subject to hear only the louder sound. He is unaware of the presence of the sound of lesser loudness if frequencies are identical. The reason that this efficient masking method is not used clinically is that patients confuse stimulus and masking. It is also well known that a low frequency sound not only masks sounds near its frequency but that masking extends to frequencies well above it. This was the basis for the use of a 60 cycle A.C. hum as a masking source in earlier audiometers. It is also one of the principles underlying the current use of complex saw tooth masking. White (random) noise is a mixture of all frequencies in approximately equal proportions. The idea underlying its use is that no matter what frequencies are tested, the masking effect should be the same. Since the effective portion of the masking intensity is only a small segment of the total at and on both sides of the frequency tested in the case of pure tone audiometry, much of

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the total intensity is useless for masking. Only a small portion known as the critical band surrounding the test frequency is effective.

In the clinical situation where a masking sound is applied to one ear to occupy or eliminate it while testing the opposite ear no effect is observed on crossed hearing as measured by bone conduction at the opposite ear until a level of 50 db re .0002 dynes/cm² is attained.³ This applies to white noise delivered by standard receivers to a normal ear in a reasonably quiet room. This level would be lower in a highly sound treated room. Under the same conditions, crossed masking occurs whenever the intensity is raised by an amount equal to the attenuation of the skull plus the ineffective component of the mask. This level for white noise is about 100 db re .0002 dynes/cm² (Table IV).³

TABLE IV

J. J. T. (Bone Conduction - Normal Subject)

250	500	1000	2000	4000	
-5	-5	-15	-5	0	No mask
0	-10	-10	0	0	Opp. ear masked 100 db re .0002 dynes/cm ²
5	0	-5	0	5	Opp. ear masked 110 db
15	10	10	5	10	Opp. ear masked 120 db

While 100 db of white noise applied to the opposite ear does not appreciably affect the threshold of the ear under test, intensities above this produce crossed masking.

Masking should ordinarily not be necessary in air conduction measurements unless the difference between the two ears is greater than 40 db. Zwislowski¹ has shown that the attenuation across the skull is about 40 db for low tones, 55 db for the middle frequencies, and 60 db for higher frequencies. The attenuation across the head for white noise is 40 to 50 db.¹⁰ This means that signals or masking sounds delivered to one ear will be heard at intensities of 40 to 60 db less in the opposite ear. Crossed hearing is largely by bone conduction and the attenuation is produced in the transfer of vibrations from air to bone. Less important factors are air conduction around the head and transmission through the handband. Once sound causes the skull to vibrate, there is relatively little further attenuation, regardless of where the sound source is applied. This is important to remember

when testing by bone conduction. It makes no difference where the bone conduction oscillator is applied; only the threshold of the ear with the better bone conduction under conditions of the test will be measured.

In case the better ear has a conductive deafness, the worse ear may show a shadow curve that is less than 40 db different by air conduction. If the conductive lesion equals the attenuation across the head, the readings may be the same. This situation is not clarified by bone conduction measurements performed either with or without masking. Tables I, II, and III show the ineffectiveness of masking in the presence of a conductive lesion.

The requirements for eliminating a normal ear by masking are fairly well established. The nonrecruiting perceptively deafened ear probably will respond to masking in the same manner as a normal to similar absolute intensities.³ A recruiting ear is probably more sensitive to masking because of abnormal loudness in the first minutes of the test and because of adaptation after this.¹¹ It is certain that the conductively deafened ear does not respond to masking intensities as does the normal. The sound intensities required to eliminate a normal ear with white noise (100 db re .0002 dynes/cm²) are at a level approaching cross masking.³ Greater intensities would cause masking of the ear being tested (Table IV).

Studies on experimentally induced conductive deafness utilizing wax plugs failed to corroborate the *a priori* idea that masking is proportionate to the degree of conductive deafness when a standard intensity of masking is employed. No relationship could be established between response to masking and degree of conductive deafness.⁸ This also seems to hold for middle ear disease as in Tables II and III where it could be proved by the combination of a dead ear on one side and a conductive lesion on the other. This indicates the need for further study to determine masking effects of commonly employed masking sounds in the surgically important conductive hearing losses and the necessity of improving present masking methods.

Dial settings of masking intensity on commercial audiometers are very confusing. In the first place, manufacturers imply and many clinicians believe that masking is uniform for all frequencies and independent of the type of deafness tested. Calibration of air conducted pure tones by periodic sound pressure readings in accordance with American Standards (American Standards Association) and frequent subjective checking on normal subjects is well established. Like-

TABLE I

O. H.

	RIGHT						LEFT					
	250	500	1000	2000	4000	8000	250	500	1000	2000	4000	8000
Air	55	70	65	65	95	95	-10	-10	-5	5	60	55
	80	95	95	95	95	95	AS masked at 100 db re .0002 dynes/cm ²					
							10	20	30	45	95	95
							Wax occlusion A.S.					
Bone	15	15	5	35	60	No mask	20	15	15	15	60	(c)
	-5	-10	-10	25	60	No mask	15	20	15	15	60	(d)
						Receiver on opp. ear						
	30	40	55	60	60	Opp. ear masked 100 db	20	25	10	15	55	(e)
						re .0002 dynes/cm ²						(g)
	0	10	15	40	60	Opp. ear occluded and						
						masked at 100 db						

B. C. readings are uncorrected machine readings.

This subject has a complete nerve deafness and absence of vestibular responses in the right ear due to complications of mastoiditis. Air conduction audiometry shows a shadow curve (a) which disappears when the left ear is masked. (b). Bone conduction measured without masking shows essentially symmetrical hearing. (c). The effect of simple receiver occlusion is dramatically shown by the improvement in hearing when bone conduction is tested over the dead ear with the good ear occluded by a receiver. (d) Note that occluding the dead ear produces no significant change.³ When the opposite ear is masked at 100 db re .0002 dynes/cm² of white noise the dead ear shows losses at or near the limits of the audiometer while the good ear shows insignificant changes. (e). The good ear was now tightly occluded by wax (flents). The audiogram (f) shows the degree of conductive deafness produced. When bone conduction at the dead ear was now tested with the good left ear masked, the excellent bone conduction shown at (g) was obtained in the low frequencies even though the conductive deafness is minimal.

TABLE II
K. B. Aged 34

	RIGHT						LEFT						
	250	500	1000	2000	4000	8000	250	500	1000	2000	4000	8000	
AIR	65	75	70	55	55	55	30	40	30	25	35	20 (a)	
AIR	70	75	90	85	75	80	A. S. masked at 100 db re .0002 dynes/cm ²						(b)
AIR	A. S. occluded						50	70	60	50	50	40 (e)	
BONE	-5	5	0	10	25	No mask	0	10	0	5	0	(c)	
BONE	10	10	40	40	45	Opp. ear masked 100 db	5	5	5	5	5	(d)	
BONE	10	10	15	25	40	Opp. ear masked 100 db	A. S. occluded						(f)

This subject has a complete nerve deafness in the right ear as shown by no hearing with Barany apparatus in A. S. and absent vestibular response and a moderate conductive deafness due to a large central perforation and residuals of otitis media in the left. Air conduction audiometry shows a shadow curve (a) which does not disappear when the left ear is masked (b). Bone conduction measured without masking shows essentially symmetrical hearing (c). When the opposite ear is masked at 100 db re .0002 dynes/cm² of white noise the left ear shows negligible changes. The right ear, however, shows an appreciable masking effect for the three high frequencies and minimal masking for 250 and 500 cps (d). When an added conductive deafness is induced by occlusion of the left ear (e), the effect on the bone conduction audiogram in the presence of the same masking intensity is to improve crossed hearing for the higher frequencies (f).

TABLE III

	RIGHT						LEFT							
	250	500	1000	2000	4000	8000	250	500	1000	2000	4000	8000		
Air	70	80	70	80	80	---	Opp. ear masked at 100 db re .0002 dynes/cm ²	50	55	50	35	45	45	(a)
BONE	5	10	10	30	30	No mask		-5	-5	5	30	30		(b)
BONE	5	10	10	35	30	Opp. ear masked 100 db		-5	-5	0	30	25		(c)
BONE	20	30	20	40	35	Opp. ear masked 120 db		0	10	20	30	30		(d)
DISCRIMINATION AD 86% no mask (Pb. at 100 db) 6% A.S. masked at 110 db re .0002 dynes/cm ² re normal threshold														
SPEECH RECEPTION		A.D. 66		A.S. 42										

This is a case of otosclerosis with postoperative labyrinthitis following a fenestration operation on the right ear resulting in a total loss of hearing in that ear. Maximal cold caloric stimulation showed no response on the right. By air conduction there is a shadow curve which persists even when the conductively deafened left ear is masked (a). The bone conduction findings are unchanged by masking at maximum acceptable intensities. (b) (c). When greater intensities are used there is overmasking as shown by the reduced threshold of the left ear. This overmasking is shown as a similar reduced threshold in the right ear. (d) Speech reception shows only 24 decibels difference between the two ears and the unmasked discrimination score at the dead right ear is quite good. When the left ear is masked at nearly equal intensities, this good hearing disappears showing that it originated in the left ear.

wise, calibration of bone conduction by testing normals if very quiet facilities are available or preferably by making a correction chart based on findings in pure nerve deafness cases as advocated by Carhart should be routine in clinical practice. The dial readings of masking sound, however, have no absolute reference levels in the majority of audiometers and few clinicians know what they are using.

Most machines are calibrated on a purely arbitrary basis. Dial settings differ widely with different manufacturers. For example, 100 db re .0002 dynes/cm² intensity corresponds to the following dial settings: Audiovox 7B 60, Sonotone SPD 80, Maico 20.

The spectrum also is not specified. Furthermore, the various dial readings on masking noise do not correspond to the decibel scale. For example, two standard audiometers read as follows:

AUDIOVOX 7B		MAICO	
(Measured in 6 cc coupler)		(Manufacturers Data)	
DIAL READING	DB RE .0002 DYNES/CM ²	DIAL READING	DB RE .002 DYNES/CM ²
50	84	10	90
60	100	20	102
70	105	30	104
80	120	40	111
90	118	50	115
		60	118

It would seem that it is high time we had a uniform standard of masking as we have for air conduction audiometers preferably prescribed by an organization similar to the American Standards Association. A standard measurable reference and a dial calibrated in db above this level would do much to end this confusion.

In the meantime, clinicians using various audiometers would be advised to know the intensity of their masking signals preferably by calibration on an artificial ear. Lacking this, manufacturers' data can be obtained or the intensity can be loudness balanced against the 1000 cycle pure tone to obtain an approximation.

If we are to know exactly what we do clinically, masking intensities will have to be spelled out. Until such time as a standard is adopted, literature on masking should state the reference level and indicate the spectrum of the noise employed. Only in this way will experiences be comparable.

Various formulae for masking have been proposed. Lateralization of the Weber is often relied upon to determine whether an ear should be masked.⁹ Reliability of this subjective test is notoriously poor; the patient tends to give answers that seem logical to him rather than to accurately describe his sensations. Furthermore, lateralization is not common in long standing asymmetrical deafness and often changes with intensity and frequency. Skulls are not homogeneous and resonant areas may influence lateralization as well as unequal ambient noise masking effects.

The point of crossover masking is an absolute function independent of the sensitivity of the ear to be masked and can be predicted approximately by adding the attenuation of the head for the stimulus to the ineffective portion of the stimulus. In the case of white noise, the ineffective portion of the stimulus using conventional head phones is about 50 db re .0002 dynes/cm² for the speech frequencies. For 250 cps the ineffective portion is 15 db higher.^{2,3} The attenuation of the head for white noise is 40 to 50 db by A.C. and negligible for B.C.¹⁰ once the sound is in the skull.

For example, when white noise is applied to an ear at an intensity 120 db re .0002 dynes/cm² the effect at the opposite ear can be computed by adding the attenuation of the head (say 50 db) to the ineffective portion of the stimulus (50 db for speech frequencies) = 100 db. Since intensities over this must be considered as capable of masking the ear under test, there will be 20 db of cross masking in this case (Table IV). This is true whether the unmasked ear is tested by either air or bone conduction.

Recognition of the short comings of commercially available masking sounds to eliminate a conductively deafened ear should stimulate the development of more effective methods. Indications of areas to be explored are the use of narrow band sounds of various configurations⁷ and the use of insert receivers instead of conventional phones to deliver the masking stimulus. It has been shown that cross masking is proportional to the area of application of the sound.¹ By applying the sound to a small area as with insert type receivers, higher intensities may be delivered to the ear to be masked without producing cross

masking. By using narrow bands of noise the ineffective portion of the total stimulus should be reduced. Another modality to explore might be the efficient Stenger principle by using the same sound for masking and stimulus but varying one by intensity modulation to permit differentiation.

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REFERENCES

1. Zwislocki, J.: Acoustic Attenuation Between the Ears. *J. Acoust. Soc. Amer.* 25:752-759 (July) 1953.
2. Harbert, F., and Sataloff, J.: Clinical Applications of Recruitment and Masking. *Trans. Amer. Laryng., Rhinolog. and Otol. Soc.*, pp. 1-14, 1955.
3. Harbert, F.: Masking Levels for Clinical Use. *A.M.A. Arch. Otolaryng.* 66:214-222 (Aug.) 1957.
4. Hazardous Noise Exposure. Air Force Regulation No. 160-3 (29 Oct.) 1956.
5. Denes, P., and Naunton, R. F.: Masking in Pure Tone Audiometry. *Proc. Roy. Soc. Med.* 45:790 (Nov.) 1952.
6. Roach, R. E., and Carhart: A Clinical Method for Calibrating the Bone Conduction Audiometer. *A.M.A. Arch. Otol.* 63:270 (Mar.) 1956.
7. Everberg, G.: Studies on the Bone Conduction in Occlusion of the Meatus and in Chronic Sound Conduction Deafness. *Acta Otolaryng.* 43:517-525 (Dec.) 1953.
8. Harbert, F., Sataloff, J., and Lerner, S.: The Effect of Masking on Induced Conductive Deafness. *A.M.A. Arch. Otolaryng.* 66:584-87.
9. Markle, D. M., Fowler, E. P., Jr., and Moulouguet, H.: The Audiometer Weber Test as a Means of Determining the Need for and Type of Masking. *Amer. Ot. Rhin. and Laryng.* 61:888-900 (Sept.) 1952.
10. Burgemeester, A. J.: Auditory Masking in Continuous Audiometry. *Acta Otolaryng.* 43:506-516 (Dec.) 1953.
11. Hood, J. D.: Fatigue and Adaptation of Hearing. *Brit. Med. Bull.* 12:2:125-130 (May) 1956.

A TECHNIQUE FOR USING
PRESERVED AUTOGENOUS SKIN
IN OTOLOGIC GRAFTING

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Re-evaluation of skin grafting techniques in surgically-created temporal bone cavities has forced itself to my attention for two reasons. First, as years of follow-up treatment of primarily grafted cavities go by, numerous initially dry ears break down with secondary infection of the skin lining. At first, the Texas Gulf coast climate was blamed. But the same thing apparently occurs elsewhere in the United States, according to informal conversation with others (Shambaugh, Walsh, and House). The second factor is the changing concept of reconstructive otologic surgery for hearing conservation sparked by the work of Moritz,¹⁷ Zöllner,^{25,26} and Wullstein.²⁴ Progress continues as others^{2,10,12,15,16} continue work with the principles of tympanoplasty and myringoplasty. The days of the classical radical and modified radical mastoidectomies as well as large cavity fenestrations are numbered. However, in my opinion, the use of preserved autogenous skin for delayed grafting has a place in our present day armamentarium.

The idea of reporting a technique for using preserved skin resulted from follow-up care of bilaterally fenestrated cases. A primary split thickness skin graft was used in one ear and a delayed autogenous graft of preserved skin was used in the opposite ear of these cases. Almost invariably the primarily grafted ear was the one that required treatment most often for secondary infection; the ear with the delayed graft would continue dry and uninfected. Biopsies of the two ears in such cases gave no clue as to why the delayed graft resisted infection.

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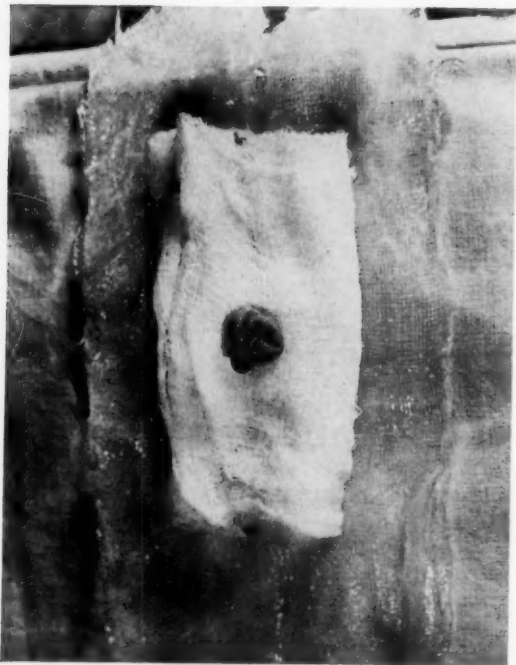


Fig. 1.—Balled up split thickness graft with raw surface innermost. Normal saline gauze and then vaseline strips enclose the ball for preservation.

Wentscher²⁰ in 1903 first reported the successful autografting of refrigerated skin. Since then there have been numerous studies of successful refrigeration of skin at various temperatures. Allgower and Blocker,¹ using tissue culture techniques, showed skin simply refrigerated in vaseline gauze to be viable for 14 days. However, Webster¹⁹ observed that skin refrigerated for 21 days would take satisfactorily. Wolstenholme²³ stated that skin preserved at ordinary ice box temperatures in a sterile jar has been found viable up to four weeks by simple refrigeration. Super cooling of skin at -70° centigrade kept it viable for 13 months according to Skoog.¹⁸ To insure consistent takes, the two weeks' viability period proven on tissue culture by Allgower and Blocker was chosen as the time for the delayed grafting in the present series. This time element would be in keeping with the recommendation of Guilford and Wright.¹³

They advocated a secondary operation under local anesthesia for cutting and placement of the graft on the tenth to eighteenth postoperative day, "as a nice firm layer of granulation tissue usually lines the cavity by this time." They feel that secondarily grafted cavities have better resistance to later infection.

METHOD

The split thickness skin graft is cut from the anterior aspect of the thigh with a Blair knife and the donor site given the dry postoperative treatment as previously described elsewhere^{21,22} in the literature by the author. When possible, the ear and thigh receive a "24-hour prep." The graft is cut before the ear surgery to avoid any cross contamination.

As soon as cut, the skin is rolled up, raw side inward, to preserve the tissue juices. The resulting ball (Fig. 1) of skin is rolled up in a sterile normal saline sponge which is then surrounded with a wrapping of two inch sterile vaseline gauze strips. This mass is tied with a black silk suture and suspended in a sterile sealed jar which has normal saline covering the bottom to insure a moist atmosphere (Fig. 2). Storage is at average refrigerator temperature (approximately 40° F.) for the two-week postoperative period. The jar is labeled with the date, patient's and physician's name, and a notation "Do not open till _____ (supply the date of the delayed grafting)." This expedient insures against accidental contamination or loss. Cultures of the preserved graft at the end of two weeks have so far failed to show bacterial growth.

At the end of two weeks, the patient's sterile head dressing is left off, the skin about the operated ear surgically prepared, and the graft skin unrolled and backed with antibiotic-impregnated rayon. Using sterile gloves, the operator then places the strips into the cavity to cover the low bed of granulations on the raw surface. No cavity packing or head dressing is applied. No premedication or local anesthesia is required for the patient.

The technique described can be applied to any surgical cavity of the temporal bone; it is simple and requires no special equipment.

Before tympanoplasty replaced classical radical and modified radical mastoidectomies in the author's surgery, this grafting method was used successfully in both the latter types of operations. Adaptations to tympanoplasty cavities was a logical step. Presently, in

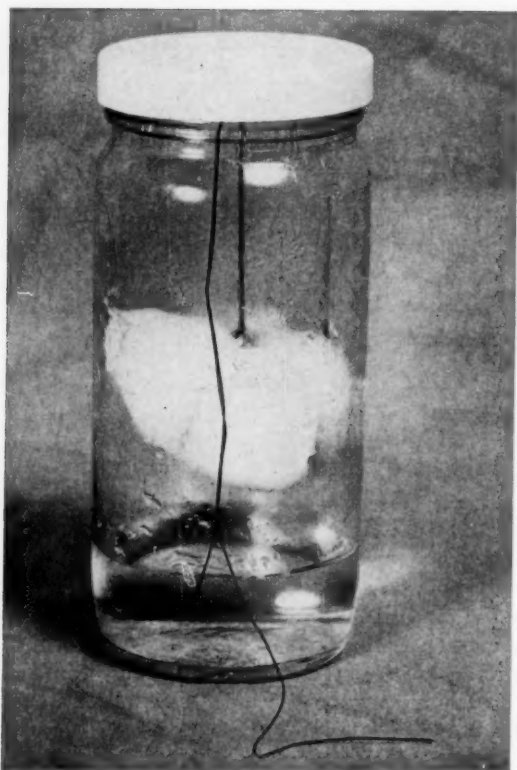


Fig. 2.—Wrapped graft skin (as seen in Figure 1) suspended in sterile jar over normal saline.

tympanoplasty, the "flap", being full thickness of tympanomeatal skin, is used as an immediate graft in one of the following ways: first, as a pedicle graft to seal off the epitympanum; or secondly, as a free graft to seal off the hypotympanum, and the eustachian tube and to offer "sound protection" to the round window; or thirdly, to close a residual drum perforation in the form of a myringoplasty. For these uses anyway, full thickness graft skin is preferred. Skin placement follows microscopic eradication of infected soft tissue and bone at the initial surgery. The triangular piece of skin from the Lempert endaural incision is placed as an immediate full thickness graft into the mastoid tip area. This skin has cerumenous glands

which help to maintain normal cavity pH to resist infection. Two weeks postoperatively, uncovered areas are lined with rayon backed, preserved skin graft strips.

In fenestration cases, a similar approach to secondary grafting is employed. Mastoid bone chips are used to fill the tip area if the cavity is large. These are covered by the full thickness triangle of skin, the tympanomeatal skin flap covers the fenestra, and the cavity is packed in the usual way with sea sponge. Two weeks postoperatively, the uncovered areas are lined with the secondary split thickness graft.

In three cases of facial nerve decompression (two temporal bone fractures, one unresolved Bell's palsy) through endaural approach, delayed grafting was used successfully in the cavities. The downward course of the decompressed nerve was covered by the tympanomeatal flap: immediate coverage of the decompressed nerve with full thickness skin in this manner did not impair return of facial function. The triangle full thickness skin piece left from the initial incision was placed in the tip after bone chips filled the excess space. To conserve hearing, the drum was packed against the capitulum of the stapes (myringostapedopexy¹⁶ or collumellar effect). In spite of this use of immediate full thickness grafts, raw, uncovered areas of the cavity remained. These were covered at the end of two weeks with the preserved autogenous skin strips.

COMMENT

Thirty surgically created temporal bone cavities were treated as described above: preserved autogenous split thickness skin was used two weeks postoperatively (in the office or clinic without anesthesia or premedication) to line the raw surface. The incidence of early dry ears was about the same as previously reported²² for immediate grafting: from four to seven weeks. However, the striking thing was how much more resistant the secondarily grafted cases were to intercurrent infection later. This finding was especially dramatic in bilaterally fenestrated cases having immediate graft in one cavity and delayed graft in the other.

REFERENCES

1. Allgower, M., and Blocker, T., Jr.: Viability of Skin in Relation to Various Methods of Storage. *Texas Reports on Biology and Medicine* 10:3, 1952.
2. Anthony, W. P.: Skin Graft Repair of Large Perforation after Fenestration. *A.M.A. Archives of Otolaryngology* 65:43 (Jan.) 1957.
3. Bradley, F. R., and Brown, J. B., Fryer, M. P., and Zaydon, T. J.: Skin Bank Stores Post-Mortem Homografts. *Hospitals J.A.H.A.* 31:54 (Jan.) 1957.
4. Brown, J. B., Fryer, M. P., and Lee, Milton: Post-Mortem Homografts as 'Biological Dressing' for Extensive Burns and Denuded Areas. *Annals of Surgery* 138:4:618-630 (Oct.) 1953.
5. Brown, J. B., and Fryer, M. P.: Post-Mortem Homographs to Reduce Mortality in Extensive Burns. *J.A.M.A.* 156:12:1163-1166 (Nov. 20) 1954.
6. Buchanan, F. T., and Lehman, E. P.: An Experimental Study of Preservation of Skin Grafts by the Freeze-Drying Process. *Surgical Forum*, pp. 637-642, W. B. Saunders, Co., Philadelphia, 1953.
7. Conway, H., Sedan, J., and Stark, R. B.: Observations on the Development of Circulation in Skin Grafts. IX. Effect of Antihistaminic (Histadyl) on Homologous Skin Grafts. *Plastic and Reconstructive Surgery* 14:417, 1954.
8. Dogo, G.: Survival and Utilization of Cadaver Skin. *Plastic and Reconstructive Surgery* 10:1:10-13 (July) 1952.
9. Farrior, J. Brown, and Iglesias, R. G.: The Primary Skin Graft in Ear Surgery: The Post-Operative Care of Mastoid and Fenestration Cavities. *Trans. Amer. Acad. of O. & O.* 59-73, 1955.
10. Gesselsson, Lennart: Improvement of Hearing by Surgical Repair of the Drum and Ossicular Chain. *Acta Otolaryngologica Supp.* 115-118:100-105 (June) 1954.
11. Georgiade, N., Peschels, E., Georgia, de R., and Brown, I.: A Clinical and Experimental Investigation of the Preservation of Skin. *Plastic and Reconstructive Surgery* 17:267, 1956.
12. Goto, Shuji: Middle Ear Operation for the Purpose of Improvement of Hearing. *Acta Otolaryngologica* 45:239-348, 1955.
13. Guilford, F. R., and Wright, W. K.: Secondary Skin Grafting in Fenestration and Mastoid Cavities. *Laryngoscope* 64:626 (July) 1954.
14. Hemphill, J. E., and Brown, J. B.: Skin Storage in Tissue Banking. *Plastic and Reconstructive Surgery* 14:2:118-125 (Aug.) 1954.
15. House, H. P.: Surgical Repair of the Perforated Ear Drum. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 62:4:1072-1077 (Dec.) 1953.
16. Juers, A. L.: Preservation of Hearing in Surgery for Chronic Disease. *The Laryngoscope* 64:235-251 (April) 1954.
17. Moritz, W.: Verschiedene Trommelfellfunctionen unter veränderter Mittelohrverhältnissen. *Arch. Ohren-, Nasen-, Kehlkopfhk.* 159:364, 1951.
18. Skoog, Tord: An Experimental and Clinical Investigation of the Effect of Low Temperatures on the Viability of Excised Skin. *Plastic and Reconstructive Surgery* 14:5:403-416 (Dec.) 1954.

19. Webster, J.: Refrigerated Skin Grafts. *Ann. of Surg.* 120:431, 1944.
20. Wentscher, J.: Ein weiterer Beitrag zur Überlebensfähigkeit der menschlichen Epidermeszellen. *Deutsche Ztschr. f. Chir.* 70:21, 1903.
21. Withers, B. T., Dickson, J. C., and Wattleworth, K. L.: Primary Split Thickness Skin Grafting of Radical Mastoid Cavities. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 61:3:656 (Sept.) 1952.
22. Withers, B. T.: Primary Grafting of Fenestration and Mastoidectomy Cavities. *A.M.A. Archives of Otolaryngology* 63:248-251 (Mar.) 1956.
23. Wolstenholme, G. E. W., and Cameron, Margaret P.: *Preservation and Transplantation of Normal Tissues.* Little, Brown and Co., Boston, 1954.
24. Wullstein, Horst: Theory and Practice of Tympanoplasty. *The Laryngoscope* 66:1076 (July) 1956.
25. Zöllner, Von F.: Die Schalleitungsplastiken. *Acta Oto-laryngologica* 44: 370, 1954.
26. Zöllner, Fritz: The Principles of Plastic Surgery of the Sound Conducting Apparatus. *Jour. of Laryngol. and Otol.* 69:637 (Oct.) 1955.

XXVII

STUDIES ON THE OXIGRAPHIC MEASUREMENT
OF THE OXYGEN TENSION
IN THE LABYRINTH

PRELIMINARY REPORT

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AND

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The basic problem of the effects of oxygen want upon bodily functions has received an ever-increasing amount of attention in recent years. A part of this problem is the behavior of the ear and more particularly, the action of the cochlea as revealed in its electrical potentials.

The author¹ came to the conclusion that the entropy production in the labyrinth is proportional to the oxygen tension. This consideration was, theoretically, derived from experimental studies of the metabolic process in the labyrinth. Now, the author's attention was called to the methods that are applied to the determination of physically dissolved oxygen in labyrinthine fluid.

Polarographic analysis was originated by Heyrovsky about 35 years ago. It is based upon the observation that when an electrolyte solution is electrolyzed in a cell composed of a dropping mercury electrode and a second non-polarizable electrode, a current-voltage curve is obtained which is characteristic of both the nature and concentration of the electro-oxidizable or electroreducible substances present. The basic theory and the limitations of the method have been extensively studied by Kolthoff and Lingane and are reported in their monograph.² Factors often neglected in quantitative polarography have been discussed by Kolthoff. The adaptability of the polarograph to the study of oxygen was utilized by Vitek;³ he found it suitable for determination of the concentration of oxygen in technical gases.

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Ingols⁴ has used the method to measure the oxygen content of activated sludge, and Manning⁵ has determined oxygen concentration at various depths in lake water. Petering and Daniels⁶ have reported the special adaptability of the technique to the study of photosynthesis and the respiratory rates of micro-organisms.

The polarographic determination of oxygen has several advantages over classical methods, the most important of which are its accuracy at low concentration and its ready adaptability to small samples. The author's concern has been to apply any method to the determination of the oxygen content of labyrinthine fluid. Unfortunately, the polarographic method for measuring the quantity of dissolved oxygen is unsatisfactory in this case, because of the very small quantities of labyrinthine fluid available, or due to the complicated structure of the labyrinth.

On the other hand, the amperometric method⁷ is a specialized growth of polarography and is often based on direct use of the polarograph. The introduction of the rotating platinum electrode for the dropping mercury electrode⁸⁻¹⁰ was a major step in making the procedure more practical. The vibrating platinum electrode¹¹ makes this technique practical for very small volumes and, therefore, a tool for microgram analysis.

Davis and Brink¹² described two types of stationary platinum micro-electrode by means of which local oxygen tensions in animal tissues can be determined with a spatial resolution of 25 μ . In one electrode the end of a platinum wire is recessed inside a cylindrical glass tip; this instrument may be used to measure absolute oxygen tensions as often as once every five minutes. In the other electrode the end of the wire is directly exposed to the other medium; with this electrode, relative oxygen tensions may be recorded continuously. An idea of the applicability of the method may be gained from the work of Davis and Brink, who employed the electrodes for the measurement of the oxygen tension at the surface of superficial arterioles and venules of the cat cerebral cortex, in the cortical substance, at the surface of muscle cells, and at chosen distances from the surface of unicellular organisms.

The principle of the method is based on the reduction at a platinum electrode of the dissolved oxygen, according to the following equation: $2H^+ + O_2 + 2e \rightarrow H_2O_2$ and the measurement of the currents developed when suitable potentials are applied, the currents being proportional to the oxygen tension.

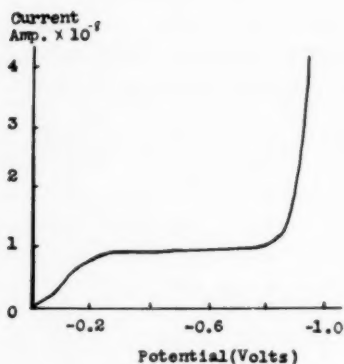


Fig. 1.—Current-voltage curve for recessed electrode in air-saturated 0.15 M NaCl. (From Davis and Brink, 1942)

The variation in electrode current with applied potentials is illustrated in Figure 1. The currents in the plateau region of the curve are limited by the maximum rate at which oxygen can diffuse to the cathode, and this rate is proportional to the oxygen tension. Also, the effect of change in the applied potential is a minimum in the plateau region and, accordingly, it is in this region that the potential are chosen for the measurement. The increase in the current above 0.8 volts is due to a second reaction, i.e., reduction from ionic to molecular hydrogen.

An instrument, i.e., the oxigraph, embodying the same principle, is now commercially available. The adaptability of this oxigraph to the study of oxygen tensions in animal tissues was utilized by several investigators.¹³⁻¹⁵ Figure 2 shows the wiring diagram for the oxigraph.

INSTRUMENT AND ELECTRODES

The platinum electrode is made from the glass tubing, which is 30 to 40 cm long and 3 mm inside diameter. Entrapped air bubbles, which may be visible only as streaks, sometimes occur in the glass tubing. It is important that the glass used for the platinum electrode has no entrapped air, which would affect the equilibrium with oxygen dissolved in the solution; accordingly, the glass should be tested by

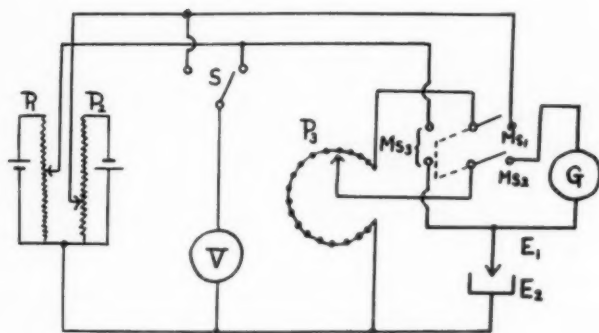


Fig. 2.—Line diagram showing the electrical circuit used cell; G , a galvanometer used for measuring the current produced; E_1 , a platinum micro-electrode; E_2 , an Ag-AgCl electrode; P_1 , an adjustable resistance used to standardize the voltmeter (V) at $+0.5$ volt; P_2 , an adjustable resistance used to standardize the voltmeter (V) at -1.0 volt; P_3 , a calibrated resistance which controls the quantity of voltage applied to electrodes.

fusing one end of the glass tubing into a ball and examining it for air bubbles.

The glass tubing is heated and, in the median region of this tubing, a slight pull is applied to give a capillary having a bore of 0.3 to 0.5 mm. A platinum wire (100 μ diameter) is introduced into the capillary, heat is applied with a microflame and the capillary tubing is pulled out to give the electrode. The outside diameter of the tip of the electrode is about 200 μ .

To avoid gas bubbles in the seal it is essential to degas the platinum before the sealing by flaming it to white heat. Before each experiment, the tip of the platinum electrode should be cleaned by flaming it to white heat to expel any gases absorbed in the surface of the tip. And the complete electrode is annealed at ca. 400 C. to prevent formation of cracks, which would cause electrical leakages.

The Ag-AgCl electrode is prepared by introducing the silver wire (1 mm in diameter), coated with silver chloride, into the soft-glass tube of comparable inside diameter, the tip of which is capillary (1 mm diameter) and filled with Ringer-agar. Then air-saturated Ringer solution is infused into the glass tubing in such a fashion that the silver wire is completely dipped in Ringer's.

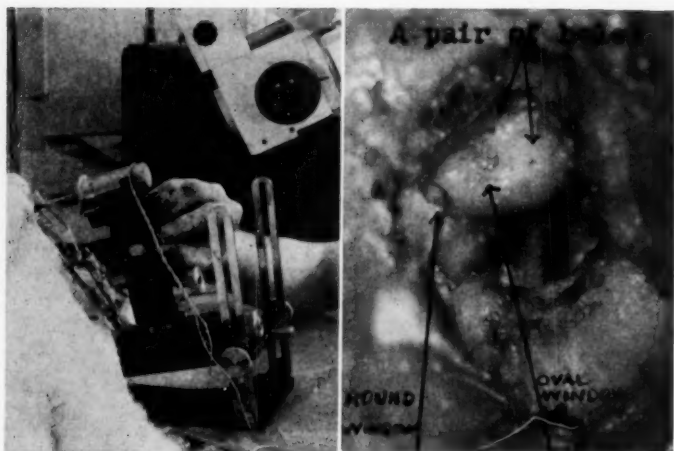


Fig. 3.—A pair of small holes are made with the aid of a microdrill mounted on the micromanipulator under the binocular microscope. The left photograph shows the microdrill. The right shows the pair of small holes around the oval window.

METHODS

The rabbits are maintained under Isozol (5-allyl-5-[1-methyl-butyl]-2-thiobarbiturate) anesthesia, supplemented with curare, and artificial respiration is used throughout the experiment.

An incision is made through the bone of the skull before the ear. An extensive area of the bone is laid bare and the tympanic bulla is exposed. The bulla is then opened with a small curet and as much of the bone removed as is necessary to provide a good view of the oval window. With the aid of the binocular microscope, the incus and the malleus are removed and the attic is opened. This sometimes necessitates the removal of the bony septum to the middle ear.

In this way, the passage is very simple. But it gives less visibility and fewer working possibilities on the stapes and the oval window. A better view of the oval window area is obtained by removing the facial nerve, with microseparators and microscrapers. Vigorous manipulations should be avoided as they can result in destruction of the bony semicircular canal or in luxation of the stapedial footplate. (This incident occurred sometimes in the author's experience; it re-

sulted in a flow of perilymph from the labyrinth.) If necessary, the head of the stapes is resected to facilitate more detailed maneuvers. If the relief of the bony labyrinthine capsule is too prominent, it is possibly rectified by a few touches of a small dental burr.

Two small holes are made at the most suitable place close to the oval window; the one in the superior position, the other in the anterior position (Fig. 3). To facilitate these direct maneuvers, a special instrument is used, a microdrill (devised by Dr. N. Maruyama), the extremity of which is 200 μ in diameter and can turn 6,000 revolutions per minute.

Small pieces of fine cellulose fiber packing, moistened with agar-gelatine-gel made by dissolving 1 g of agar and 5 g of gelatine in 100 ml 0.9% sodium chloride solution, are then held on the superior hole to acquire thereby a firm barrier between the middle ear and the perilymphatic space. The tip of the platinum electrode is inserted into the anterior hole about 0.1 mm in depth from the internal bony wall, so as to attach to the surface of the membranous labyrinth (Fig. 4), and the reference electrode (Ag-AgCl electrode) is placed in the packing described above (Fig. 5). The tip of the platinum electrode is just adjustable to the hole and therefore the outflow of the perilymph through the hole (with the electrode in) is practically insignificant. Then the middle ear is filled completely with liquid paraffin and, therefore, the contamination of air with perilymph is practically negligible.

RESULTS

According to the oxigraphic studies of the labyrinths of the rabbit, the authors have carried out the measurement of the oxygen tensions in the labyrinth which revealed deleterious effects upon the labyrinth as a result of anoxia.

For example, a small degree of anoxia does not cause a significant change of oxygen tension in the labyrinth (Fig. 6). But longer periods of anoxia cause the increase of oxygen tension and the oxygen tension recovers to an initial level by rebreathing (Fig. 7).

Of course, the oxygen tension in the labyrinth decrease in additional periods of anoxia. On the other hand, the cutting experiments on the common carotid artery revealed that no change of oxygen tension is evident under conditions in which the animals are seriously affected, and up to the point where a severe loss of blood puts an end to observations.



Fig. 4.—The Pt-electrode is inserted in the anterior hole.



Fig. 5.—Ringer-agar-gelatine-gel packing is placed on the superior hole, and is pressed by the capillary tip of Ag-AgCl electrode. This electrode is fixed on the micromanipulator.

COMMENT

The basic problem of the effects of oxygen want upon bodily functions has received an ever-increasing amount of attention in recent years. One of the recent advances in biophysics of the labyrinth, the studies of the cochlear microphonics, opens a new field of research, explaining many complicated physiological procedures.

The effects of oxygen deprivation upon cochlear microphonics have been investigated repeatedly and invariably lead to the same conclusion, namely, that cochlear microphonics are not affected until oxygen deprivation becomes so severe that breathing and circulation begin to fail.

In order to discuss the possible significance of these findings, it is necessary to estimate the oxygen tension in the labyrinth during exposure to oxygen lack. In the field of otology, an understanding of this problem is the great frontier to be conquered.

The estimation of the oxygen content of bodily fluids by means of polarograph was described by Beecher et al.,¹⁶ but the method of Davis and Brink would appear to offer the best opportunity for the application of the polarographic technique to respiration on a scale for histochemical work. Davis and Brink,¹² Clark,¹⁷ and Mochizuki¹⁴ measured the oxygen tensions in the cerebral cortex of the cat. In the latter experiment, the electrodes were arranged in the bony skull as shown in Figure 8.

But these technical procedures restrict its application in the study of the labyrinth quite severely, either because of the small size of the labyrinth or the minimum volume of intralabyrinthine space available, or due to the complicating structure of the labyrinth. The author's method appears to have been successful in the measurement of oxygen tension in the labyrinth, while preserving the labyrinthine functions. According to Davis and Brink,¹² the variable properties of the open electrode make for considerable instability in its performance. Calibration before and after each experiment will hold the error to the minimum. Poorly-defined plateaus are obtained in current-potential curves, but as a rule with up to 0.8 volt nothing but oxygen is electrolyzed in oxygen-free solution. A linear relation may be found between current and oxygen tension in calibration experiments, however, when applied to tissue; particularly near blood vessels, there may be little correlation between the actual tension and that derived from a calibration curve which is obtained with a

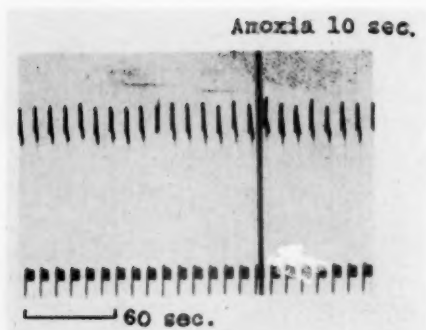


Fig. 6.—The anoxic effects on oxygen tension in the labyrinth.

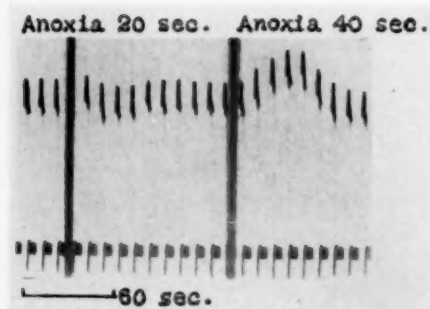


Fig. 7.—The anoxic effects on oxygen tension in the labyrinth.

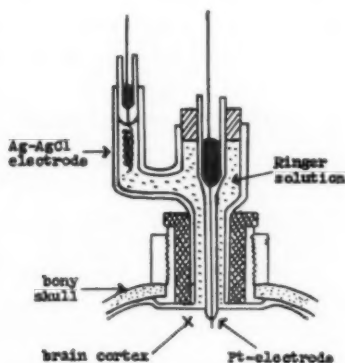


Fig. 8.—The arrangement of electrodes for the measurement of oxygen tension in the cerebral cortex of the cat. (From Mochizuki, 1953)

solution such as Ringer's. There is, then, a limit to the usefulness of this method, because the open electrode cannot be used for the measurement of absolute oxygen tension, and because the response time per stage cannot be reduced less than approximately ten seconds, so far as this oxigraph is concerned.

The platinum electrode, as mentioned above, is inserted in a hole which is made close to the oval window with the aid of the microdrill, so as to attach its tip to the surface of the basal turn sac. However, the main problem in such a procedure is the considerable instability in the actual position of the tip of the electrode due to the variability of the anatomical structure of the labyrinth.

The authors wonder whether its tip attaches always to the surface of the membranous labyrinth. It is reasonable to surmise that the current curve obtained in the experiments does not always reflect the change of oxygen tension of the endolymph, but the change of oxygen supply by the labyrinthine vessels.

Previous investigations revealed the high resistance of the cochlear microphonics to anoxia. No doubt a number of physiological conditions are involved, but it is evident that one of the important factors is oxygen starvation of the cells in which the potentials are generated.

The present study on the oxygen tension in the labyrinth suggests this fact. Although our observations are not ideal for this purpose, still these results provide the basis for a preliminary view.

SUMMARY AND CONCLUSIONS

Oxygraphic measurement of oxygen tension in the labyrinth (rabbit) has been carried out by the authors with the aid of special instruments. This current curve obtained reflects the change of oxygen supply by the labyrinthine vessels.

As anoxia develops over about 20 seconds, the oxygen tension in the labyrinth undergoes a rapid initial increase and, with extreme anoxia which carries the animal close to the point of death, then tends to level off.

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REFERENCES

1. Koide, Y.: Introductory Studies of Chemical-Physiology of the Labyrinth. *Jibi Rinsyo* 50:377, 1957.
2. Kolthoff, I. M., and Lingane, J. J.: Polarography, Interscience Pub. Co., New York, 1941.
3. Vitek, V.: Collect. Czechoslov. Chem. Communicat. 7:357, 1953 (Cited by Beecher et al.)
4. Ingols, R. S.: Dissolved Oxygen Recording with the Dropping Mercury Electrode. *Ind. Eng. Chem., Anal. Ed.*, 14:256, 1942.
5. Manning, W.: *Ecology* 21:509, 1940 (Cited by Beecher et al.)
6. Petering, H. G., and Daniels, F.: The Determination of Dissolved Oxygen by Means of the Dropping Mercury Electrode, with Application in Biology. *J. Am. Chem. Soc.* 60:2796, 1938.
7. Laitinen, H. A.: Amperometric Titrations. *Anal. Chem.* 21:70, 1949.
8. Kolthoff, I. M., and Pan, Y. D.: The Amperometric Titration of Lead with Dichromate or Chromate. *J. Am. Chem. Soc.* 61:3402, 1939. The Titration of Sulphate and Some Other Anions with Lead and the Reverse Titrations. *J. Am. Chem. Soc.* 62:3332, 1940.
9. Laitinen, H. A., Jennings, W. P., and Parks, T. D.: Amperometric Titrations of Chloride, Bromide, and Iodide Using the Rotating Platinum Electrode. *Ind. Eng. Chem., Anal. Ed.*, 18:358, 1946.
10. Kolthoff, I. M., and Harris, W. E.: Amperometric Titration of Mercaptans with Silver Nitrate Using the Rotating Platinum Electrode. *Ibid.* 18:162, 1946.

11. Kirk, P. L.: Quantitative Ultramicroanalysis. John Wiley and Sons, Inc., New York, 1950.
12. Davis, P., and Brink, F.: Microelectrodes for Measuring Local Oxygen Tension in Animal Tissues. *Rev. Sci. Instruments* 13:524, 1942.
13. Mochizuki, M.: The Measurement of Oxygen Tension in the Living Body. *Medical Science* 3:212, 1952.
14. Mochizuki, M., and Kirikai, H.: On Blood Flow and Oxygen Concentration in Brain Cortex During Convulsions. *Respiration and Circulation* 1:229, 1953.
15. Mochizuki, M., and Bartels, J.: Amperometrische Messung des Oxygen Druckes in Vollblut mit der blanken Platinelektrode. *Pflügers Arch.* 261:152, 1955.
16. Beecher, H. K., Follansbee, R., Murphy, A. J., and Craig, F. N.: Determination of the Oxygen Content of Small Quantities of Body Fluids by Polarographic Analysis. *J. Biol. Chem.* 146:197, 1942.
17. Clark, L., Wolf, R., Granger, D., and Taylor, Z.: Continuous Recording of Blood Oxygen Tensions by Polarography. *J. Appl. Physiol.* 6:189, 1953.

XXVIII

VOCAL BREAKDOWN ITS CAUSE AND CURE

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That the voices of many outstanding singers deteriorate long before their possessors give evidence of a general decline of their physical powers is a fact that seems to be accepted as inevitable. The acceptance of this fact points to the deplorable lack of understanding of the underlying causes for this decline, which, once they were properly understood, would permit singers to pursue their careers undisturbed, and afford their audiences the opportunity to enjoy their art when it has reached full maturity. As the matter now stands, all too often when a singer has amassed the necessary experience to be at his best, he finds his vocal powers have declined to a point where he can merely indicate what he would like to do, but is not able to sing with the freedom and power which were once a part of his performance. In other words, when he has reached the pinnacle of his artistry, his voice no longer responds to his wishes. While he may still have many devoted followers who go to hear him and applaud him, yet in whispered asides they express their sorrow that his voice is no longer what it used to be.

The familiar explanation is that the singer has sung for so many years that it cannot be expected that his voice could still be as good as it formerly was, as the wear and tear on the vocal organ is too great. It does not seem to be realized that the declining artist is still able to walk and talk without difficulty and is often able to do so many, many years after his vocal career has come to an end.

This brings us to the question: what is the difference between singing and talking, since both are produced by the very same organ? The major difference is that singing requires a wider range of pitch and somewhat of an increase in volume. Pitch changes are brought about by the action of the internal muscles, notably the thyroarytenoids with some assistance from the palatopharyngeus and stylopharyngeus. These latter are particularly concerned with the production of the higher tones. If these muscles be given proper exercise there

is no valid reason why they should deteriorate any earlier than other muscles of the body. But since, in the decline of vocal powers, it is the higher tones which first begin to evidence weakness, it is reasonable to assume that, as with all muscles, inadequate exercise must be at the root of the trouble. It will, of course, be argued that a singer who is singing actively makes steady and continuous use of his vocal muscles so that there should be no reason for weakness to develop. Taken at its face value this argument is logical so that the true cause of the muscular deterioration must be due to the employment of a method of voice production which deprives the vocal muscles of their proper exercise.

The suggestion that a singer who has attained prominence has been using his voice incorrectly will, of course, be vehemently protested, but it is of such singers that we are speaking. It is their performances that the public and critics find lessening in appeal so that, despite all arguments to the contrary, it must be possible to achieve success even when the voice is not being used correctly.

Many years ago, Clarence Lucas, during the time he was associate editor of the *Musical Courier*, made the following statement regarding singers which I feel can be of great help in understanding this seeming paradox. He said: "Many a young singer does all sorts of foolish things with his voice with hardly a protest from the vocal cords and no warning at all from the music critics. The freshness of the young voice lasts long enough for the bad habits to become fixed. And then the bloom begins to wear off the peach." This may serve to explain why it is not at once evident that the voice is not being used correctly and there are, of course, many degrees of incorrect production. However, even a small degree of tension gradually increases the difficulties which beset the singer, until finally these become too great to permit of satisfactory singing.

It would therefore appear that the singer belongs in a very different category from the instrumentalist, for instrumentalists are often able to maintain their ability to perform long after maturity has been reached. But instrumentalists perform upon instruments which are constructed for the purposes for which they are used. The singer, however, as is now beginning to be recognized, makes use of an organ the true function of which is not that of producing tone. In addition to this, misuse of the vocal organ begins practically at birth so that long before any attempt is made to make use of the organ for professional purposes bad habits are deeply ingrained and should first be eradicated before proper use of the organ can be achieved. Unlike

the instrumentalist, the singer does not ever start with a clean slate. Even if the amount of his singing has been negligible there are bad habits of speech to consider and these must be overcome before real progress can be expected. It has been falsely assumed that singing is a natural means of expression, but the fact that the vocal organ was not designed for the production of tone negates this belief. While it is true that some people seem to sing more naturally and easily than others, this difference is one of degree, not of kind. There is invariably some degree of interference with the free action of the vocal organ and even if the amount be ever so slight, if the singer is not taught how to detect it and then eliminate it, it will inevitably increase to a point where it becomes a threat to his singing.

The idea that the sole requirements for the teaching of singing consist of a voice, a good ear and some knowledge of music should have been abandoned long ago. Singers, unfortunately for them, are incapable of hearing their voices exactly as the listener hears them, and are therefore dependent upon outside assistance. So that if those who offer this assistance are uninformed as to the manner in which tones are produced and ignorant of the factors which prevent tones from being freely produced, the singer is truly leaning upon a broken reed. That such methods do not produce satisfactory results is evidenced by the fact that singers wander from studio to studio seeking the help they so badly need, only too often ending up in complete disillusionment. So far as the teaching of singing is concerned, no discernible standards exist, and it is futile to expect valid rules of procedure to be developed unless all those who are teaching possess a similar technical background. As Clarence Lucas said, fresh young voices sound well even if the tones are not being correctly produced, so that it would appear necessary to have more information about the production of the voice than that supplied by the ear. It seems logical to believe that without a sound technical understanding of the actions and interactions of the parts of the throat which are concerned in the production of tone, the teacher is not capable of providing accurate information as to how a pupil can best eliminate his faults. As was said earlier, it is not a question of making use of an instrument specifically designed for the production of tone. The teaching of singing should consist (aside from the musical values involved) of instructing the student how to detect and eliminate those factors which hinder him in the development of his vocal organ. Since the vocal organ is subconscious in action, it cannot be consciously trained, therefore the stress must not be laid upon the vocal organ, but upon those parts of the throat which inhibit its freedom of action without which its development cannot successfully be undertaken.

The singer who is unable to find a logical and rational answer to his problems faces an inevitable decline of his vocal powers. Since it takes a good many years to learn all the facets of vocal expression, only too often by the time he has, what might be called, "learned his trade," he finds that all the finer points which he has learned to make use of in his singing can no longer be achieved. Instead of a fine functioning organ which once seemed to be his, he is left with an organ which he neither can cajole nor force to do his bidding. The more he relies upon the powerful external muscles to produce tone, the weaker the internal muscles become until a point is reached where he is obliged to abandon all attempts to sing. This condition can rightfully be described as a true vocal breakdown. And, strange as it may seem, this condition is not the result of overuse or overstrain of the vocal muscles, but is due to a weakening caused by lack of proper exercise. There is but one cure for this condition and it can be brought about by first eliminating the outside help the organ has been receiving and then exercising the internal muscles until they recover their strength and elasticity. Such an undertaking requires a great deal of patience and tenacity of purpose, but once it has been accomplished the singer finds himself in full possession of his vocal powers, in addition to which he has at his command all the skill and experience in interpretation which had been acquired during his singing career.

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XXIX

SURGERY FOR ADVANCED
EAR CANCER

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Cancer of the ear has been divided into the intrinsic type, which involves the middle ear and mastoid, and the extrinsic type which is located laterally to the ear drum. For practical purposes this subdivision may be of value in the management of small lesions located externally to the ear drum, where conservative surgery will permit wide excision without sacrifice of the important structures. However, this classification is no guide in determining the treatment of extensive cancer involving the middle ear and surrounding bone. The site of origin is often obscure, and only the most radical measures will salvage the patient. Unfortunately, by the time the majority of cancers of the ear canal and middle ear are diagnosed, there is gross involvement of the middle ear, mastoid, petrous bone and temporomandibular joint. Roentgen diagnosis is almost never made until the very late stages of the disease. Except for an occasional primary mastoid cancer, the majority of cancers involving the middle ear represents an advanced stage of the disease. Most of the patients presenting at Memorial Center with this disease have had a mastoidectomy and x-ray therapy, and have had a recurrence. Our effort has been directed towards salvage, by en bloc resection of the cancer-laden bone with the temporomandibular joint and adjacent soft tissues. The purpose of this paper is to discuss an experience with 27 cases of ear cancer treated by temporal bone resection during the past five years.

Furstenberg¹ estimated the incidence of cancer of the middle ear at 1:20,000 of all aural conditions. At the Mayor Clinic² the incidence was reported at 0.03 per cent of all cancers over a six year period. Lodge et al.³ found six cases in a population of a million. These

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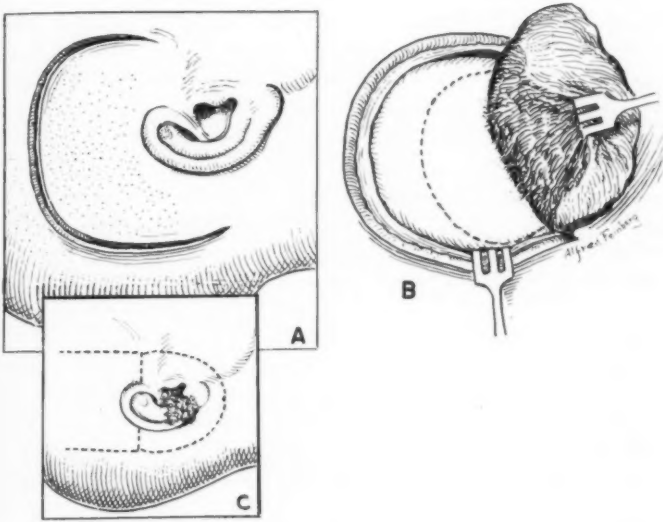


Fig. 1.—Types of incision. A and B, preserving the pinna; C, for excision of the external ear with the specimen. (The illustrations are from a previous paper by the authors, published in *Cancer*, Sept. 1954.)

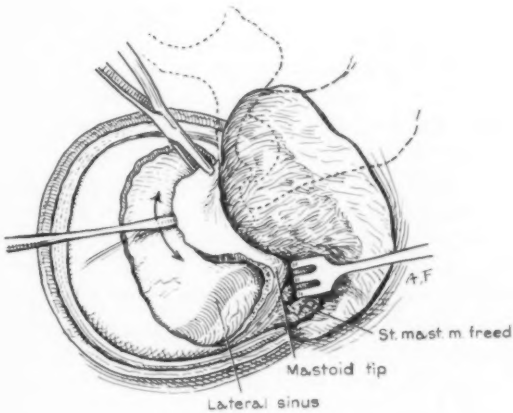


Fig. 2.—Removal of the squamosa of the temporal bone.

authors felt that cancer in the ear is comparable to a Marjolin ulcer, a malignant ulceration in cutaneous scars and chronic ulcers with sanious discharge producing intracellular irritation and subsequent cancer growth. In the six cases described by these authors chronic otorrhea was the precursor of cancer development. Several of our cases developed on this basis, others were associated with chronic otitis externa, and several had no apparent antecedent pathology.

Of 27 cases operated upon to date, 21 cases were squamous carcinoma; three cases were basal cell carcinoma; two cases were sarcoma (one spindle cell sarcoma and one embryonal rhabdomyosarcoma); one case was a malignant mixed tumor of the parotid gland.

Of the 27 cases presenting at the hospital, 12 were primary in the middle ear; 12 others appeared to originate in the external auditory canal; one case was primary in the mastoid; one case appeared to originate in the pinna and extend through into the ear canal, middle ear and mastoid; and one case was primary in the parotid gland and extended through the base into the middle ear.

The patients treated varied in age from six years to 75 years; the median age was 55 years.

Of the 27 cases treated, 18 occurred in females and nine in males. Of the females, nine originated in the middle ear, eight in the external auditory canal, and one in the parotid gland. Of the males, three occurred in the middle ear, four in the external auditory canal, one in the mastoid and one from the pinna. There appears to be a predilection for cancer of the ear canal and middle ear in females in a ratio of 2:1 and 3:1 in this series.

The symptoms of cancer of the ear, pain, bleeding and aural discharge, and perhaps facial nerve paralysis, are those of advanced cancer. Lesions arising from the external ear and eroding into the middle ear and mastoid are obvious. Those arising in the ear canal and middle ear are sometimes confused with chronic inflammation, and biopsy diagnosis marred by the presence of granulation tissue. Perhaps cytologic examination by the Papanicalaou method may uncover the cancer process earlier. Deep-seated parotid cancer may erode the floor of the ear canal and middle ear and simulate a primary lesion. Cancer of the soft palate and nasopharynx may extend up the eustachian tube into the middle ear. Clinical examination of the entire head and neck region is important, and careful palpation for cervical metastases is mandatory. The presence of bone destruction

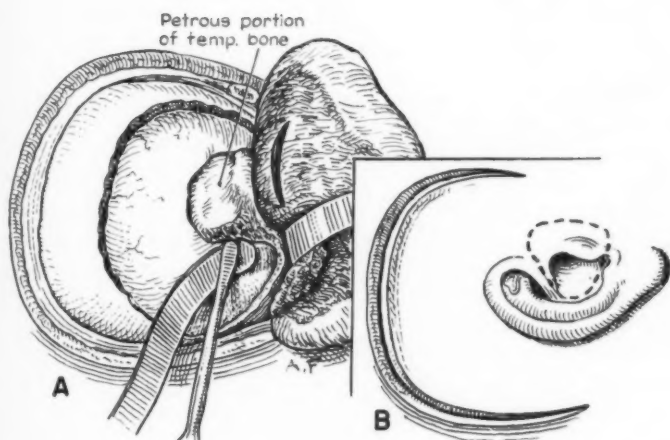


Fig. 3.—A, freeing the dura from the petrous ridge and, with B, incision for coring out the external auditory canal.

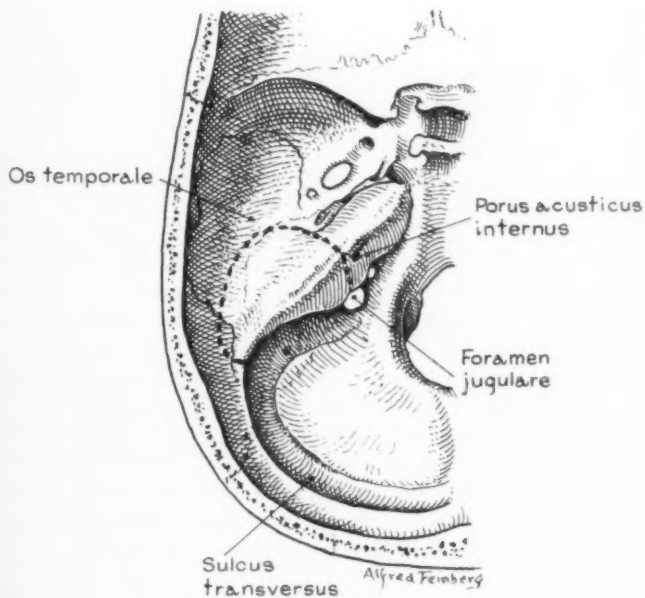


Fig. 4.—Extent of bone removal of the petrous ridge.

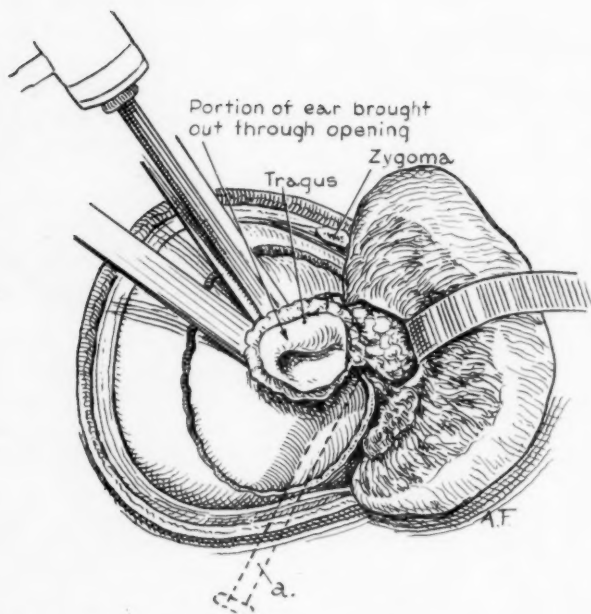


Fig. 5.—Placing of chisels for fracturing the petrous portion of the temporal bone.

on x-ray is often an omen that cancer has extended beyond the bony confines of the temporal bone, although often the actual extent of the disease cannot be determined until operation. A negative x-ray does not in itself augur well, for a sclerotic mastoid may mask disease.

OPERATIVE PROCEDURE

The technique of the temporal bone resection has been described by the authors previously.⁴ The approach is a combined temporal craniotomy with exposure of the dura in the region of the temporal lobe of the brain. Cerebrospinal fluid is removed through a previously inserted malleable spinal puncture needle and the dura and brain mobilized from the petrous portion of the temporal bone. The lateral sinus is elevated from the operative field and the superior petrosal sinus retracts with the dura. The ascending ramus of the mandible and the temporomandibular joint and base of the zygoma are excised along with the specimen. The petrous portion of the temporal bone

is fractured at the junction of the medial and middle thirds. With the use of chisels carefully placed the en bloc specimen with the soft tissues of the upper neck region and mastoid come away with the specimen. Bleeding from the lateral sinus is not infrequent during the procedure but usually can be controlled by gelfoam and pressure or by the use of a silk suture with or without temporal muscle tampon. If the dura is involved it can be excised and replaced by temporal fascial graft. The defect is skin grafted with a split thickness graft and a firm cornish wool stent applied to the excision site. Large defects have been covered by a pedicle scalp flap (Figs. 1 to 7).

Fortunately complications have been few considering the magnitude of surgery. There were three deaths in the postoperative period. One occurred suddenly on the tenth postoperative day and autopsy could not be obtained. This patient had vagus paralysis requiring tracheostomy and pulmonary complications may have been the cause of his exitus. Another death occurred on the fourteenth postoperative day from meningitis and cerebral softening. A third patient died four weeks after surgery with thrombo-embolic phenomena secondary to a bacterial endocarditis. The organism involved in this latter case was staphylococcus. Several patients have had cerebrospinal fluid fistulae resulting from a dural tear and drained intermittently for varying periods up to three months. However, all these fistulae closed spontaneously. In eight other cases a portion of the graft sloughed. In two other cases there was transient postoperative cerebral edema following ligation of the lateral sinus. In four cases tracheostomy was necessary because of vagus nerve paralysis with laryngeal incompetence. However, in the majority of the cases (except for the expected facial and acoustic nerve paralysis) neurological complications have been few.

Of the 27 cases operated upon since this procedure was first performed, 18 cases appeared to be completely operable. No gross residual disease was evident following temporal bone resection. In nine cases of the 27 operated upon, there appeared to be residual disease involving the brain and the base of the skull following removal of the temporal bone specimen. These latter cases were considered incurable by surgery in that residual disease was left behind. All of these nine cases received postoperative irradiation, including betatron and cobalt therapy.

The first case subjected to temporal bone en bloc resection at Memorial Center was in 1951, so that a five-year cure rate for this procedure is not possible at this time. At present eight patients are

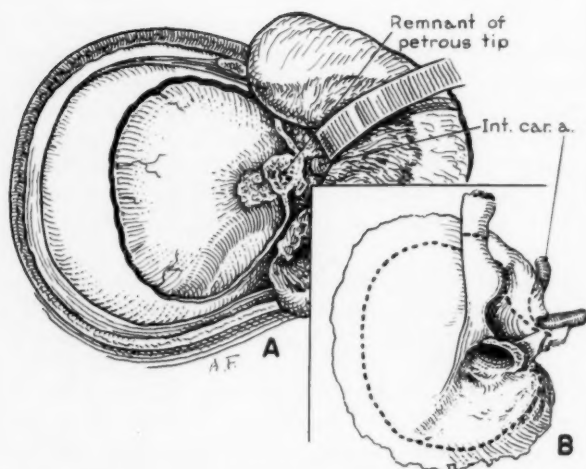


Fig. 6.—A, the operative field following removal of the major portion of the temporal bone; B, extent of bone removal, lateral view.

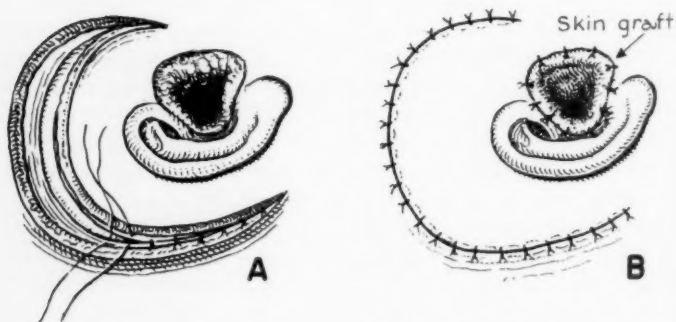


Fig. 7.—A, closure of the wound; B, closure with skin graft inserted into the ear-canal defect.

free of disease from periods of one to five years. Six cases have gone uneventfully for more than one year; two of these for more than three years. All of these cases were primarily operable, and represent a substantial number of the 18 cases found to be operable.

The nine patients with residual cancer subjected to postoperative irradiation have all succumbed to their disease.

Three patients developed metastatic neck nodes. One case was subjected to a radical neck dissection, but recurred both locally and in the neck, and the patient expired from disease. Two other cases with positive neck nodes were irradiated. One of these cases is over one year without recurrent disease; the other patient expired from widespread local recurrence and neck metastases.

SUMMARY

During the past five years 27 cases of cancer involving the middle ear were subjected to a temporal bone en bloc resection with skin graft of the defect. The operative technique is described and the complications are mentioned. Because of the short-term experience with this operation, we have only one patient that is free of disease for five years. However, eight patients are free of disease for more than one year. The end results from the standpoint of cure and palliation with this operation appear to be most encouraging.

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REFERENCES

1. Furstenburg, A. C.: Primary Adenocarcinoma of the Middle Ear and Mastoid. *ANNALS OF OTOLGY, RHINOLOGY AND LARYNGOLOGY* 33:677, 1924.
2. Quoted by Lodge, W. O., Jones, H. M., Smith, M.: Malignant Tumors of the Temporal Bone. *A.M.A. Arch. Otol.* 61:535-541, 1955.
3. Lodge, W. O., Jones, H. M., Smith, M.: Malignant Tumors of the Temporal Bone. *A.M.A. Arch. Otol.* 61:535-541, 1955.
4. Parsons, H., and Lewis, J. S.: Subtotal Resection of the Temporal Bone for Cancer of the Ear. *Cancer* 7:995-1001 (Sept.) 1954.

Scientific Papers of the American Otological Society

XXX

THE EFFECT OF TRACTOR NOISE ON THE AUDITORY SENSITIVITY OF TRACTOR OPERATORS

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During the practice of otolaryngology for the past several years in a rural area, a sufficient number of farmers with 4000 cycle dips have been noticed to attract special attention. In some instances the ear history and physical examination have been negative except for the probability of tractor-noise induced hearing loss. However, it also has been observed that certain farmer patients who drive tractors have been seen with normal hearing.

Very little has appeared in the literature about the effect of tractor noise on auditory sensitivity. Bunch³ in 1937 presented an audiogram of a 26 year old farmer with a bilateral high frequency hearing loss, presumably due to tractor noise. Wilson⁹ in 1941 presented four audiograms of farmers who drive tractors which "... typically demonstrate cochlear damage of traumatic origin in the basilar turn," and concludes that "the tractor presents an as yet unrecognized source of occupational deafness." These audiograms by Bunch and Wilson suggest that tractor noise may be the principal etiologic factor in the production of high frequency hearing losses in tractor operators. These audiograms point out the probable existence of a specific tractor noise problem but contribute nothing to knowl-

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edge about the physical characteristics of tractor noise or the incidence of noise induced high frequency hearing impairments in farmers who drive tractors.

This paper presents the results of a two-part study of the effect of tractor noise on auditory sensitivity: the first part deals with the measurement of tractor noise; the second part is concerned with the results of pure tone threshold sensitivity measurements on 80 full time tractor operators who presented otherwise negative ear histories.

A. Measurement of Tractor Noise. Since quantitative data on the sound pressure levels (SPL) of tractor noise were not readily accessible, it was necessary that such measurements be obtained. The SPL of tractor noise was measured by a team of five participants as follows: an assistant, riding on the tractor and behind the operator, held the dynamic microphone of the sound level meter six inches laterally from first one ear and then the other of the tractor operator. A 25 foot cable extended from the microphone to the battery energized sound level meter and octave-band noise analyzer in an adjacent station wagon. While both vehicles were in motion, so that the tractor noise could be measured while the tractor was operating under normal working load conditions, a second assistant read the over-all and octave-band SPLs. A third person acted as recorder of the data and the remaining member of the team drove the station wagon. Incidentally, the experimental team was surprised and delighted with the degree of constancy and repeatability of the measurements.

B. Quantitative Measurements. Figure 1 shows the SPL contours of 14 sets of measurements made on 11 different tractors manufactured by seven different companies. As indicated above, these measurements were obtained while the various tractors were working under load conditions and while the microphone was held six inches from the ear of the tractor operator. Differences in SPL within six inches of the right and left ears were negligible while the operator was looking forward in the direction the tractor was travelling. Six tractors used diesel oil and five used gasoline as fuel; most of the diesel oil operated tractors had two cylinders and most of the gasoline operated tractors had four cylinders. The power ratings of the various tractors varied from 35 to 60 horsepower.

Two tractors had rear mufflers; the remainder had forward vertical mufflers. Measurements were obtained on one tractor with

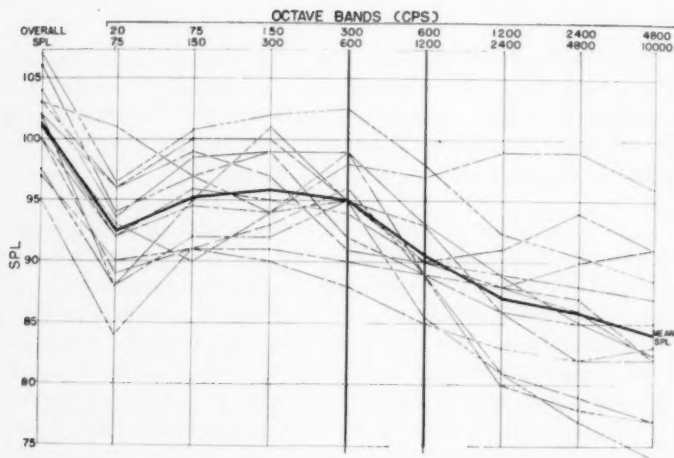


Fig. 1.—Sound pressure levels of tractor noise in db re 0.0002 dynes per sq. cm.

and without the muffler; one tractor pulled an operating experimental picker-sheller and one tractor pulled an operating production model two-row corn picker; the other tractors either pulled plows or an equivalent experimental brake load.

The decision to measure tractor noise while the tractors were operating under work load conditions appeared essential. Measurements on one of the tractors showed an over-all SPL of 100 db while working but a 97.5 SPL while in a fast idle state. The noise produced by the accompanying station wagon had no effect on the tractor noise measurements; when the motor of the tractor was stopped the over-all noise level immediately dropped to around a 60 db SPL while the station wagon was still in motion. Tractor noise measurements were obtained with the weighting network of the sound level meter set at the C (flat) position.

Figure 1 indicates that tractor noise is distributed over a rather wide frequency range, having about 10 db less pressure in the 4800-12,000 cycle band than in the 300-600 cycle band. The various

tractors and loads produced a SPL range from 88 to 102.5 db in the 300-600 cycle octave band and an 85 to 98 db SPL range in the 600-1200 cycle octave band. The heavy curve shows the mean overall and octave band SPLs.

In Figure 2 the top curve A shows the SPL six inches laterally from the ear of the operator while a tractor was pulling a production model two-row corn picker while picking corn. Curve B shows SPL measurements made on the same make and model tractor while plowing. Inspection of curves A and B shows that the tractor-corn picker combination produced a 9 db higher SPL in the 300-600 cycle band and a 4 db higher SPL in the 600-1200 cycle band than the tractor-plow combination. These A and B curves indicate that some of the attachments used with tractors generate more noise than the tractors themselves, a finding with which farmer tractor operators are familiar. Some of these attachments are constructed in part with large flat thin metal plates, which form vibrating surfaces while the equipment is working under load.

Figure 3 shows SPLs of a tractor-load combination without a muffler and with two different types of mufflers. The experimental muffler, Curve C, attenuates the SPL 8 db in each of the two critical octave bands and attenuates the noise considerably more above the 600-1200 octave band than at the lower frequency levels.

COMMENT

Does tractor noise damage hearing? Obviously, the answer to this question can be obtained most directly by testing the auditory sensitivity of individuals exposed to tractor noise. However, sufficient general information about the relationship between hearing impairment and noise exposure is available to permit certain useful predictive assumptions about the effect of noise on hearing. According to the Academy Revised 1957 Guide for Conservation of Hearing in Noise,¹ "At the present time our knowledge of the relations of noise-exposure to hearing loss is much too limited for us to propose 'safe' amounts of noise exposure. . . . This tentative hearing conservation level is stated as follows: If the sound energy of the noise is distributed more or less evenly throughout the eight octave bands, and if a person is to be exposed to this noise regularly for many hours a day, five days a week for many years, then: if the noise level in either the 300-600

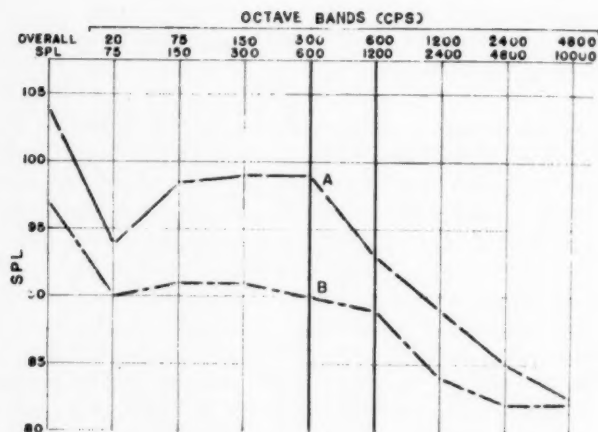


Fig. 2.—A. SPL of tractor pulling two-row corn picker. B. SPL of tractor.

cycle band or the 600-1200 cycle band is 85 db, the initiation of noise-exposure control and tests of hearing is advisable. The more the octave band levels exceed 85 db the more urgent is the need for hearing conservation."

The measurements summarized in Figures 1, 2 and 3 show that the SPLs generated by tractors under load equal or exceed the critical 85 db SPLs in the 300-600 and 600-1200 cycle octave bands. The mean SPL in the 300-600 cycle band was 95.0 db with a range extending from 88 to 102.5 db; the mean SPL in the 600-1200 cycle band was 90.5 db with a range of 85 to 98 db. The probability certainly exists that tractor noise is sufficiently high to constitute an occupational hazard. As indicated above, many of the earlier tractors were not equipped with mufflers and generated higher SPLs than the modern machines. Even today many tractor operators are careless about muffler replacement or repair.

Most of the noise generated by tractors pulling quiet loads, such as plows, comes from the exhaust or open end of the muffler. In the past, certain makes of tractors produced a high intensity level sound

which could be attributed to gear noise. However, it is possible by the use of modern precision gears to attenuate this source of noise below the level of that emanating from the exhaust. It appears plausible that tractor noise can be attenuated still more by judicious placement and design of mufflers, plus the application of vibration damping principles in the construction of certain tractor attachments such as cornpickers and shellers.

MEASUREMENT OF THE AUDITORY SENSITIVITY OF TRACTOR OPERATORS

A. *Subjects.* In order to obtain pure tone audiograms of a group of individuals who are exposed to considerable tractor noise, tests of threshold sensitivity were performed on 91 men who are full-time tractor operators on the farms of the seven adjacent Iowa Amana Colonies. During the winter season these men drive tractors attached to wagons to haul feed for livestock, lumber, fertilizer and in general perform the services required for the operation and maintenance of 25,000 acres of productive farm land.

B. *Testing Technique.* The hearing sensitivity was tested at five frequency levels in the following order: 1000, 500, 1000, 2000, 4000 and 8000 cps. The first test at 1000 cycles was regarded as introductory practice or conditioning and was not recorded. Right ears were tested first, unless the subject believed his hearing was worse in his right ear. In theory, the testing order should have alternated between the right and left ears to equate possible learning and practice effects. A tone presentation technique in which the intensity was increased progressively from inaudibility to audibility was employed. The tests were conducted in the different offices of the seven farm managers. These offices were one story wooden frame structures in which no other activity was in progress during the testing procedure. The ambient noise was surprisingly low in each of these locations since they were a considerable distance away from heavy traffic and other sources of noise. Testing was suspended when running tractors were near or an airplane passed overhead. With the exception of such infrequent noises, the background noise levels in each of the testing rooms did not exceed an over-all SPL of 40 db, using the C (flat) scale of the Sound Level Meter. The audiometer earphones were supplied with type NAF-48490-1 "doughnut" earphone sockets and mounted in a headband which held the earphone-

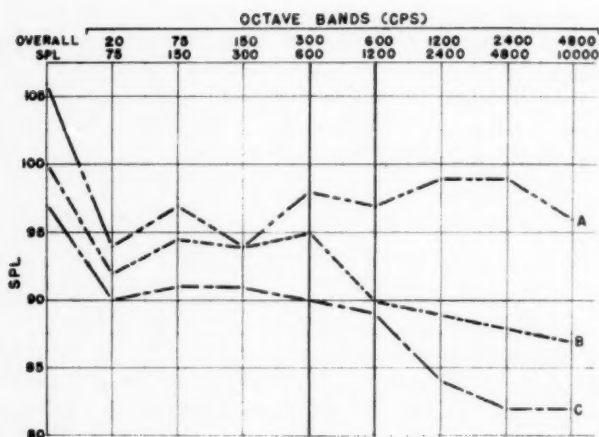


Fig. 3.—A. SPL of tractor without muffler. B. SPL of same tractor with production muffler. C. SPL of same tractor with experimental muffler.

socket assemblies with firm pressure against the sides of the head. A group of four university students exhibited no statistically significant differences in threshold sensitivity measurements obtained in a laboratory sound proof room and in five of the seven testing locations. (The students were not tested in the two remaining stations.)

Practically all of the tests were given during the middle of the week and following the lunch hour. The tests were conducted during the late summer and early fall of 1957. A pertinent history was obtained for each subject, including information relative to ear disease, tinnitus, drug therapy, occupational and military activities and the type of firearms used in hunting and target practice. The hearing sensitivity of 91 tractor operators was tested. Eleven were excluded from the present report because of hearing losses resulting from ear disease, traumatic head injury or accidental sound blasts in military service. Unilateral measurements were used in two instances; one man had had a mastoid operation on one ear with a residual tympanic hearing impairment and the other had had Ménière's disease with a resultant unilateral cochlear involvement; the opposite ears in each of these two subjects had negative ear histories. The present

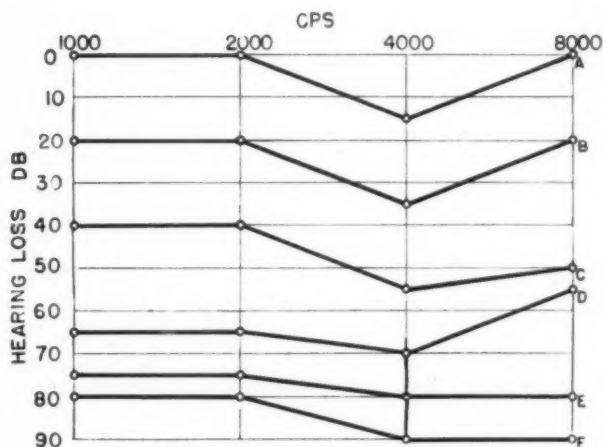


Fig. 4.—Quantification of dips. A = 15 db. B = 15 db. C = 5 db. D = 5 db. E & F = inflections or knees.

study presents threshold sensitivity measurements on 158 ears of 80 tractor operators.

C. Background for Interpretation of Hearing Tests. Bunch and Raiford,⁴ as a result of audiometric measurement of 300 white males and 224 white females in 1931 found that "In each decade low tones are heard better by the males, and high tones are heard better by the females." This greater high frequency loss in males has been confirmed by all later studies of threshold sensitivity. Not only do males have greater hearing impairments for high frequencies than females at comparable age levels but the mean sensitivity curves of males in several studies show 4000 cycle "dips" at certain age levels which are not exhibited by females.

Since the present study reveals rather large 4000 cycle dips in comparison to previous presbycusis studies, it is necessary to describe a method for the quantification of dips. The term "dip" in the present frame of reference implies a restricted frequency range for which the sensitivity is less than for bordering higher and lower frequencies. In the present paper, a 4000 cycle dip will be quantified

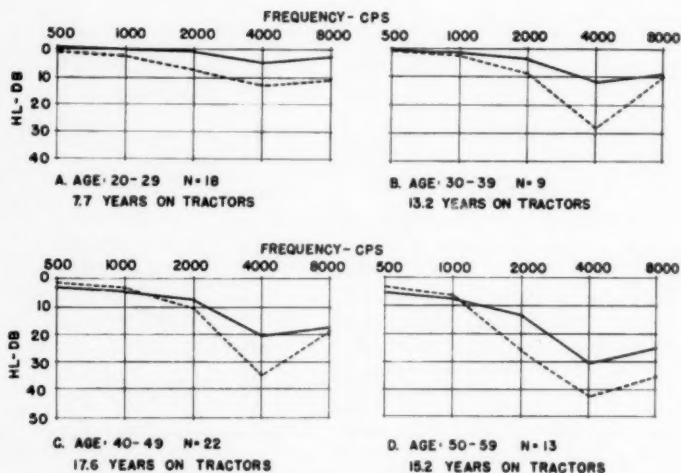


Fig. 5.—Variations in threshold sensitivity with age. Means of composite surveys ——— Means of tractor operators - - - - -

as a "4000 cycle 15 db dip" if the hearing loss is 15 db greater at 4000 cps than for the adjoining higher and lower octaves, regardless of the absolute hearing loss levels of the three frequencies. When the hearing losses differ at the adjoining higher and lower frequency levels, the 4000 cycles dip will be quantified by the db difference between 4000 cps and the adjoining octave for which there is the greater loss. For example, in Figure 4, curves A and B both show a 4000 cycle 15 db dip; curves C and D both show 4000 cycle 5 db dips, although the D type of curve is atypical; curves E and F do not show dips but a bend which is called an inflection or a "knee." A knee in this connotation may be defined as the point on a sloping curve at which the greatest change in the slope occurs.

The maximum 4000 cycle mean dips found by Bunch and Raiford,⁴ who tested 300 males, occurred in the 30-39 and 40-49 age groups and did not exceed 10 db. Beasley,² after testing the sensitivity of 2,002 males who had negative ear histories and who experienced no difficulty in understanding speech, found mean dips not exceeding 3 db in any decade of the normal hearing group. (How-

ever Beasley did find after analyzing threshold sensitivity data of 624 persons who had a clinical history of Stage I deafness that 4000 cycle mean dips as great as 8 db occurred in certain age groups. Stage I deafness was defined as "that preventing a person from understanding speech in a public auditorium—theatre or church—or in a conference between five or six people, but permitting one to understand speech in direct conversation at a distance of two or three feet.") Leiste,⁶ after testing 211 males with negative ear and noise histories, found the maximum mean dip at 4000 cycles did not exceed one db at any decade. Steinberg, Montgomery and Gardner,⁷ after analyzing the results of hearing tests on 17,079 males at the New York City and San Francisco World's Fairs, found maximum mean dips not exceeding 1.6 db at 3520 cycles within any age decade. Webster, Himes and Lichtenstein,⁸ after testing 2,112 males at the San Diego Fair did not find mean 3520 cycle dips at any age level. (The preceding two hearing surveys employed test frequencies of 440, 880, 1760, 3520 and 7040 cps.) Glorig et al.,⁵ after testing 1741 males at the Wisconsin State Fair, did not find median 4000 cycle dips at any age decade in the total sample. However, test frequencies above 6000 cps were not used in this survey. Small median dips were found between 3000 and 6000 cps in 26 male farm workers and 85 male factory workers in the 30-39 year age groups. (A median dip did not occur for 87 office workers at this same age level.) Small median dips also were found between 3000 and 6000 cps in 55 men who had been in the air force, 122 men who had been in the Army and 14 men who had been in the marines, who were in the 30-39 year decade.

The two studies mentioned above which did not disclose mean dips in the total male samples at any age decade did show knees in the curves at the 4000 cycle level at several age levels. It appears significant that 4000 cycle dips were found more consistently and pronounced at the 30-39 and 40-49 age decades. The absence of dips in the older age groups probably is due to the combined effects of noise and age at the higher frequency levels, but with the age factor predominating. It appears that presbycusis is not characterized by dips but by a gradual decrease in sensitivity to the highest audible frequencies which progressively involves the adjacent lower frequencies, encroaching upon the upper speech frequencies during the later decades. It also appears that permanent hearing impairments resulting from exposure to noise are characterized by dips, although certain

exceptions may occur. Such being the case, the presence of a dip assumes important diagnostic significance.

D. *Origin of Base-line Presbycusis Curves.* The presbycusis curves with which the auditory sensitivity of the tractor operators in the present study is compared were obtained by combining the data on 23,445 males in the six hearing surveys mentioned above. Each study was weighted in proportion to the number of subjects in each and the mean hearing losses calculated at the comparable frequency and age levels. This procedure is open to a certain amount of adverse criticism. The World's Fair⁷ hearing tests influenced the composite curves disproportionately since about eight times more males were measured at the World's Fairs than in the next largest study. Tests were conducted at both the World's Fairs and the San Diego Fair⁸ at

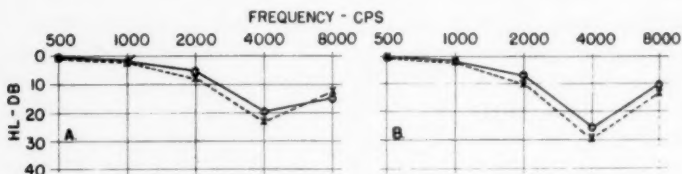


Fig. 6.—Mean threshold sensitivity of non-servicemen and servicemen. Rt. ear o-o-o- Lt. ear x-x-x- A. Sensitivity of 26 non-servicemen. Mean year on tractors: 12.4; mean age: 35.1 years. B. Sensitivity of 23 servicemen. Mean years on tractors: 13.5; mean age: 33.5 years.

440, 880, 1760, 3520 and 7040 cps, which were combined with the respective 500, 1000, 2000, 4000 and 8000 cps frequency levels used in the other studies. The test tones employed at both the World's Fair and the San Diego Fair studies were reproduced from phonograph records which placed limitations on the maximum intensity ranges at 3520 and 7040 cps. The Beasley² group of 2002 males included only individuals who stated they had no difficulty understanding speech, which undoubtedly eliminated many with moderate and severe presbycusis. About 80 per cent of the World's Fair measurements were on the left ear only while the better ear only was tested at the San Diego Fair. Most of the papers reported the data in terms of the mean but the median was used in the Wisconsin⁵ and the San Diego⁸ Fair studies.

All the studies undoubtedly included a high percentage of males who had an indeterminate amount of hearing loss which had resulted from noise exposure, in addition to that due to the presbycusis factor.

E. *Auditory Sensitivity of Tractor Operators.* The clinical audiometer used for obtaining the threshold sensitivity measurements was calibrated according to the American Standards Association and American Medical Association specifications. The calibration was rechecked twice during the testing interval and found unchanged. The output intensities of the two earphones differed by slightly less than one db at each test frequency level.

Figure 5 shows the comparison between the composite presbycusis curves (in solid lines) and the threshold sensitivity curves of the left ears of the tractor operators (in broken lines). Two differences between them are apparent: 1) the sensitivity of the tractor operators is worse above 1000 cps than that of the general population and 2) the tractor operators have greater dips at 4000 cps, especially at the 30-39 and 40-49 decades.

The Wisconsin Fair study analyzed the sensitivity of male office workers, farmers and factory employees. In general, office personnel had the least impairment, farmers had greater losses than the office workers and factory employees had the greatest loss of the three groups. The tractor operators of the present study had greater median impairments than the Wisconsin Fair office workers. However, the Wisconsin Fair farmers, in general, had somewhat greater impairments than the Amana tractor operators up to and including 4000 cps.

Sensitivity measurements of left ears only are shown in Figure 5 since the left ears, especially those above 30 years of age, had slightly greater hearing losses than the right ears. The Wisconsin Fair survey was the first to show statistically significant worse sensitivity for left ears than for right ears. Earlier surveys, with the exception of that of Beasley, were not designed in such a way that differences between the ears would become apparent. Beasley² found "that the left ears are slightly but significantly better than the right ears on most tones" in a "normal" group of 1,242 males and females whose air conduction audiograms for both ears did not exceed a variation of 15 or 20 db on his eight test tones (Bulletin 4, p. 6). In the summary (Bulletin 4, p. 18) Beasley states that the "correlation coefficients for measure-

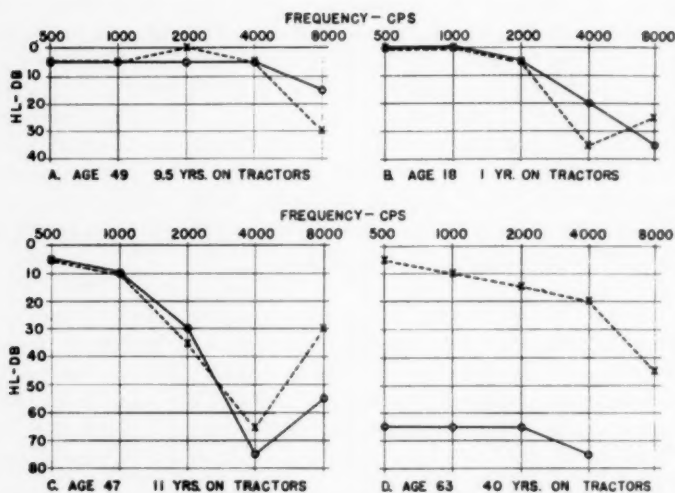


Fig. 7.—Selected audiograms of tractor operators.

ments on the right and left ears by air conduction do not vary markedly in magnitude with audiofrequency level. . . . However, the coefficients tend to be higher for tones below 1024 cycles than for tones above this level." The Wisconsin Fair data indicated that "at all frequencies the left ears of males in all age groups show more loss than the right."

Several investigators have noted that the sensitivity of the left ears of individuals who often shoot shotguns and rifles may be worse than that of their right ears. Twenty-four tractor operators who frequently fired guns and an equal number who never or rarely did so were matched in age and number of years of tractor operation as closely as possible. The mean ages of the hunters and non-hunters were 39.5 and 41.3 years respectively; the mean number of years of tractor operation of the hunters and the non-hunters were 13.8 and 12.4 years respectively. The mean threshold sensitivity of the left ears was 1 to 3 db worse than that of the right ears at 1000, 2000 and 4000 cps in each group. The differences were less at 500 and 8000 cps. The hunters in general had 1 to 2 db worse sensitivity in each ear than the non-hunters.

Since the threshold sensitivity curves of the tractor operators showed considerably larger dips than the comparable composite presbycusis curves, the data were analyzed for the incidence of dips. Forty of the 80 operators had bilateral dips of 5 db or greater, 24 did not have a dip in either ear and 16 had unilateral dips. Eleven of the unilateral dips were in left ears and five in right ears.

F. Effect of Military Service on Threshold Sensitivity. Three of the group of 91 tractor operators were eliminated from the present study because of a history of exposure to training or combat noise which resulted in subjective hearing impairments which were found to be pronounced on audiometric examination.

Twenty-three operators between the ages of 20 and 50 had been in military service and 26 within this same age span had not. Figure 6 shows the mean sensitivity of the left and right ears of each group. It is apparent that the sensitivity of both ears of the service men is somewhat worse than that of the non-servicemen, and that the sensitivity of the left ears of each group is worse than that of the right ears, particularly at 2000 and 4000 cps.

G. Individual Differences in Threshold Sensitivity of Tractor Operators. Figure 7 shows four audiograms which illustrate certain interesting features in relation to the general trends mentioned above. 'A' shows the sensitivity of a 49 year old man who had operated a tractor for 9.5 years, had not been in military service and who is an avid huntsman. His hearing is within normal variation limits for his age level and does not suggest any past occupational noise exposure. 'B' shows the audiogram of an 18 year old youth who had driven a tractor for one year and who had never hunted nor been in military service. Although his ear history is negative otherwise, it is not assumed that his hearing loss resulted from exposure to tractor noise. 'C' shows the sensitivity of a 47 year old man who had operated a tractor for 11 years, who had never been in military service and who has done a moderate amount of hunting. Comparison of audiograms 'A' and 'C' is instructive. Both men are reasonably well matched in relation to age, years of operating experience, hunting and neither was in military service. 'A' has normal hearing for his age level but 'C' shows a marked bilateral impairment of the type which could have and probably did result from noise exposure. 'D' shows

the audiogram of a 63 year old man who had driven a tractor for 40 years, had one year of military service in World War I and who has hunted very little. The measurements on his left ear do not reveal a dip and the over-all sensitivity of this ear indicate somewhat better than normal hearing for his age level. The severe impairment in his right ear resulted from Ménière's disease and was not included in the group mean data. It appears likely that this operator possesses a "tough" left ear. By contrast, operator 'C' could be said to have a "tender" ear. It is obvious that tractor noise does not impair the hearing of all tractor operators. Apparently, only those with tender ears are susceptible to threshold shifts as the result of tractor noise exposure.

H. *Tinnitus*. The histories revealed that many tractor operators with marked hearing impairments were acutely aware of tinnitus, especially at night, whereas those with unusually good hearing for their age levels usually were not conscious of tinnitus at any time.

I. *Left Ear vs. Right Ear Sensitivity*. Reasons for the greater losses in the left ears are largely speculative. The shooting of firearms is not the only explanation.

A factor which might contribute to the relatively greater loss in the left ears of tractor drivers is the fact that the operator, especially while plowing, is frequently looking over his right shoulder at the furrow, thereby directing his left ear to the open end of the tractor exhaust which usually is located on the front of the tractor.

It is interesting to note that Glorig et al⁵ also found that "for females in all age groups the left ear shows more loss than the right at the higher frequencies only; the reverse obtains at the low frequencies." It may be that the common application of the telephone receiver to the left ear and the exposure of left ears of automobile drivers to noise entering the open window on the left also contribute to the slightly greater impairment of left ears in both sexes.

CONCLUSIONS

Results of this preliminary study must be considered tentative until confirmed by a larger number of auditory measurements. Within the limitations of this study, the following trends are summarized:

1. Tractor noise is sufficiently high in intensity to produce high frequency hearing losses in tractor operators with noise susceptible ears if exposed over a period of several years.
2. The intensity of tractor noise can be reduced by the design of more effective mufflers; the noise of certain tractor attachments also can be reduced by utilization of vibration control construction methods.
3. High frequency dips are more common between the 30 and 60 year age levels.
4. The absence of dips in elderly individuals does not rule out the possibility of some noise induced hearing loss.
5. Slightly greater losses occur in left than in right ears.
6. Individuals with noise susceptible ears usually experience tinnitus. Such persons should wear effective ear plugs while operating tractors.
7. It is recommended that hearing surveys in the future be designed to test sensitivity at 500, 1000, 2000, 3000, 4000, 6000, and 8000 cps in order to obtain more information about the frequency-intensity characteristics of dips and to learn more about the aging ear.

UNIVERSITY HOSPITALS

Grateful appreciation is extended to the John Deere Research and Engineering Center of Waterloo, Iowa, for invaluable assistance in obtaining the tractor noise measurements.

REFERENCES

1. American Academy of Ophthalmology and Otolaryngology: Guide for Conservation of Hearing in Noise, 111 North Bonnie Brae Street, Los Angeles 26, California.
2. Beasley, W. C.: National Health Survey, Hearing Study Series, Preliminary Reports, 1938, Bulletins 1-7. The U. S. Pub. Health Serv., Washington.
3. Bunch, C. C.: The Diagnosis of Occupational or Traumatic Deafness; A Historical and Audiometric Study. *Laryngoscope* 47:615-691, 1937.

4. Bunch, C. C., and Raiford, T. S.: Race and Sex Variations in Auditory Acuity. *Arch. Otolaryngol.* 13:423-434, 1931.
5. Glorig, A., Wheeler, D., Quiggle, R., Grings, W., and Summerfield, A.: 1954 Wisconsin State Fair Hearing Survey; Statistical Treatment of Clinical and Audiometric Data, *Amer. Acad. Ophthalmol. and Otolaryngol.* 1957.
6. Leiste, T. J.: Audiometric Studies of Presbycusis. *Acta Otolaryngol.* 37: 555-562, 1949.
7. Steinberg, J. C., Montgomery, H. C., and Gardner, M. B.: Results of the World's Fair Hearing Tests. *Jour. Acoust. Soc. Amer.* 12:291-301, 1940.
8. Webster, J. C., Himes, H. W., and Lichtenstein, M.: San Diego County Fair Hearing Survey. *Jour. Acoust. Soc. Amer.* 22:473-483.
9. Wilson, W. H.: Occupational Deafness. M.S. Thesis, Univ. Minn., 1941.

XXXI

ANATOMICAL STRUCTURE OF THE STAPES AND THE RELATION OF THE STAPEDIAL FOOTPLATE TO VITAL PARTS OF THE OTIC LABYRINTH

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The purpose of this report is two-fold: first, to describe features in structure of the stapes pertinent to problems which confront the otologist in attempting stapedial mobilization; second, to demonstrate the relation of the footplate to the otic (endolymphatic) labyrinth, a relationship which inescapably places the labyrinthine duct-system in jeopardy in the course of endaural surgery.

MATERIAL AND METHODS

From a re-examination of more than 150 series in the otological collections at the University of Wisconsin and at Northwestern University Medical School, new or confirming data have been gathered on the architecture of the stapedial footplate in prenatal and postnatal stages. These records have been further amplified by re-examination of developmental stages contributory to the frail structure of the stapes in the adult ear (Figs. 1, 2 and 6). From series in the collection at the University of Wisconsin a reconstruction was prepared to demonstrate the relation of the footplate to the vital parts of the otic labyrinth in the vestibule (Figs. 3, 4 and 5).

OBSERVATIONS AND COMMENTS

Stapedial Architecture. In the fetus of 9 weeks the auditory ossicles, although definitely formed, are still cartilaginous. The stapes, still ring-shaped rather than stirrup-shaped, is lodged medially in

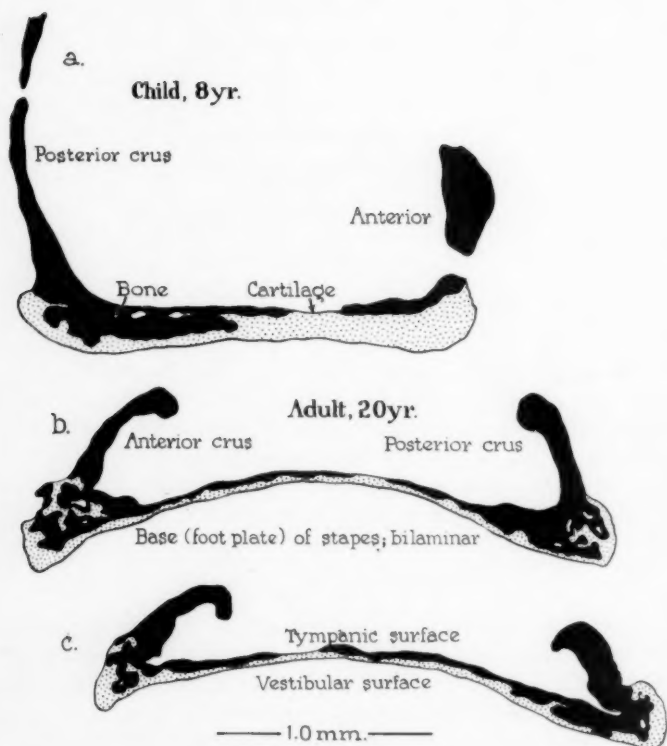


Fig. 1.—The structure of the base of the stapes, shown semischematically (from tracings prepared by Edinger projection apparatus) X 32.

Fig. 1a.—Section through one of many areas in which cartilage is exposed on the outer (tympanic) surface of the base of the stapes from a child eight years of age. Demonstrating the excessively thin base and the occurrence of areas in which the osseous lamina of the base of the stapes is wanting in small zones. Fig. 1b and 1c.—Comparable structure in the adult.

capsular tissue which now closes the area of the future vestibular fenestra (oval window).

In a fetal specimen of 20 weeks, the single ossification center of the stapes has made its appearance on the wall of the obturator space in the area of continuity of the crus with the base. In other specimens of approximately the same age, periosteal bone is present, for the first time, in the form of a pellicle externally; internal to the thin layer of bone vascular buds erode the subjacent cartilage. Ossification is still limited to the crurobasal segment of the ossicle, where the process of erosion spares the vestibular lamina of the base.

At a slightly later stage (21 week fetus) the osseous shell is circumferential for each crus; on the contrary, the vestibular lamina of the base, the entire neck and head remain cartilaginous. In the crural portion of the stapes, marrow occupies the area where cartilage formerly occurred.

In the 23-week fetus, ossification has advanced to include the neck of the stapes, spreading along the crura toward the head of the ossicle. Cartilage remains at the basal extremity, but is eroded internally; on the internal surface, endochondral bone is forming.

The stapes in the fetus of 24 weeks is composed of bone, except at the extremities; medially cartilage covers the vestibular surface and the fenestral margin, while laterally the primordial cartilage of the capital end persists as an articular plate (at the incudostapedial joint). Erosion of the periosteal layer on the obturator aspect has kept pace with formation of this external shell. Internally, spicules of endochondral bone, meager in amount, are transitorily present; they will be removed except where such bone lines the cartilaginous plate of each extremity (basal and capital).

The stapes of the 32-week fetus, now of adult form and structure, is completely invested by mucous membrane. Both the basal and capital extremities are bilaminar, each of the layers being thin but complete.

All of the structural features which characterize the stapes in the late fetus and in the newborn infant are retained in virtually unaltered form in the adult (Figs. 1 to 3). The ossicle is excessively thin; its

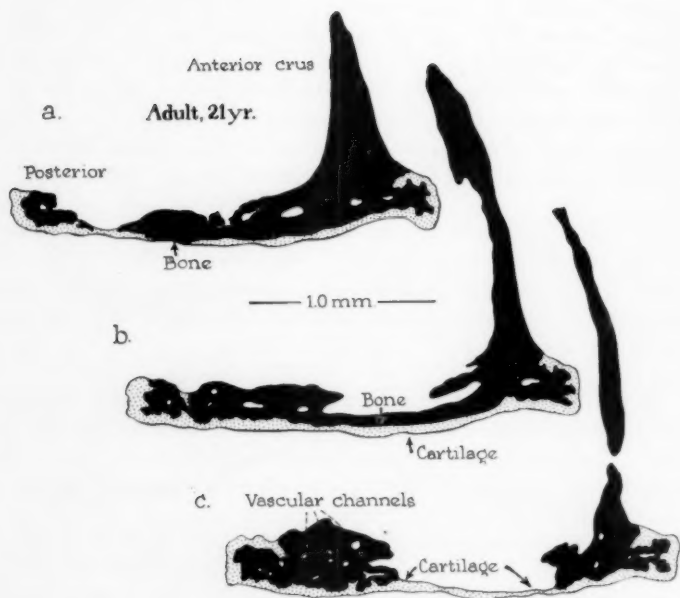


Fig. 2.—Structure of the stapedial base, continued. Adult 21 years of age. X 27.

Fig. 2a.—Demonstrating the thickening of bone which regularly occurs in the area of continuity of a crus with the base (at the reader's right) and the thinning which is characteristic of other parts (at the reader's left). Fig. 2b.—An area where bone and cartilage are of approximately the same thickness except along the periphery of the base. Cartilage, in all normal ossicles, is carried from the vestibular surface to invest the base circumferentially where the base faces the wall of the vestibular (oval) fenestra. Fig. 2c.—The section includes an area in which cartilage is the only constituent (between the arrows) of the base of the stapes. Marginally bone is thick and contains numerous vascular channels.

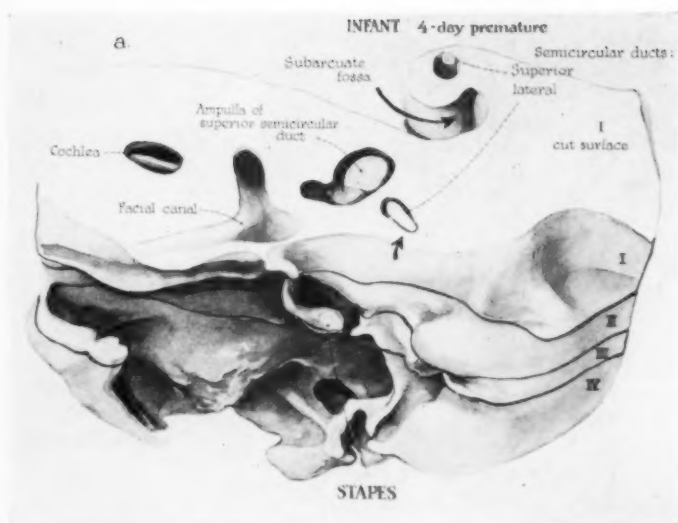


Fig. 3.—Drawing of a reconstruction of the petrous part of the temporal bone in a newborn infant (4-day premature). The reconstruction shown entire, specially to record general topographical features. The unlabelled arrow indicates the approximate site for surgical fenestration. Viewed from a lateral (tympanic) and superior (cranial) position. Left ear, X 5.

base, like the head, is two-layered (one layer being the cartilaginous lamina of the vestibulo-fenestral surface, the other being the endochondral lamina of the tympanic aspect). Mucous membrane now covers not only the outer surface of the periosteal bone of the crura and head, but also coats the inner surface of the same layer, where, as a result of removal of the obturator wall of the ossicle, the mucosal tunic comes to replace the primitive marrow and the endochondral spicules. The mucous membrane on the base of the stapes lies in contact with the endochondral lamina, which, in a process of secondary growth, formed a covering for the persistent cartilage.

No exception to this type of stapedial architecture was encountered in the course of examination of 150 otological series recently re-examined.

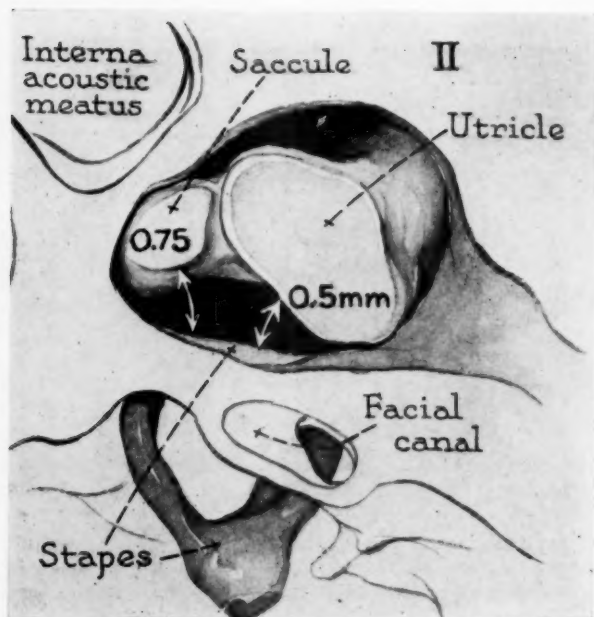


Fig. 4.—The superior surface of the second (II) segment of the reconstruction (see Fig. 3). Demonstrating the relation of the stapedial base to the utricle, saccule and the internal acoustic meatus. X 19.

Stapedial Relationships. The internal ear comprises the following: the otic labyrinth, consisting of an epithelial-lined labyrinthine cavity filled with otic (endolymphatic) fluid; the periotic labyrinth, consisting of periotic fluid filled spaces, which in part surround the epithelial otic labyrinth; the supporting connective tissues, including the basilar membrane, the organ of Corti, limbus, tectorial membrane, spiral ligament, nerves, ganglia, and the internal periosteum. All of these are surrounded by the bony otic capsule. When the above-mentioned soft structures are removed, as occurs in dried temporal bones, the space left in the otic capsule is known as the bony labyrinth.

A Woods metal cast of such a bony labyrinth presents a three-dimensional concept of the total volume of this space. Such a model

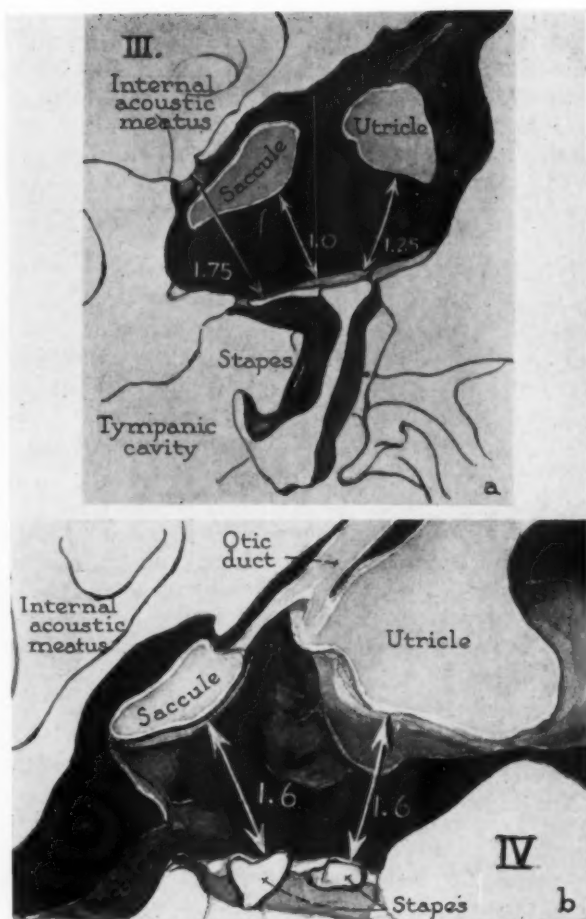


Fig. 5.—Corresponding surfaces of the same reconstruction at more inferior levels. X15. a.—Superior aspect of Segment III. Detail with measurements of distances from the vestibular surface of the base of stapes to the utricle, the saccule and the inferior vestibular area, where minute foramina transmit nerve twigs to the saccule. b.—Superior surface of Segment IV, approaching the inferior limit of the cochlea and of the vestibular window with the stapes, passing through the vestibular aqueduct and spherical recess of the vestibule.

displaces approximately two-tenths of a cubic centimeter, or about three drops of water. When space occupied by the epithelium, the neuroepithelium, the spiral organ of Corti and all the other soft supporting tissues has been deducted from this content, the total volume of fluid in the internal ear must be substantially less than the volume of water displaced by the Woods metal cast. If one further considers the fact that the two fluids, otic (endolymphatic) and periotic (perilymphatic) are contained in small capillary spaces, it becomes apparent that, due to capillary action, little or no fluid should escape when the labyrinth is opened.

The small capacity of the internal ear also indicates a circumstance commonly overlooked, namely, that the various parts of the otic labyrinth are extremely small and separated by very short distances. The most voluminous part of the internal ear is the vestibule, a somewhat flattened ovate space 6 to 7 mm in length, 4 mm in maximum diameter and 3 mm in minimum diameter.

Within the vestibule lie the sacculus and utricle. The sacculus measures about 1 to 1.5 mm in its greatest diameter, and the corresponding diameter of the utricle is about 2.5 to 3 mm. In the lower part of the vestibule the sacculus and utricle are separated from each other by about 1 mm, but in the upper part their walls are in contact.

Equally important are the spatial relationships between the stapedial base (footplate) and the various vital parts of the labyrinth. Figures 4 and 5 represent these relationships: anterior part of oval window to the sacculus, 1 mm; anterior part of oval window to the internal auditory meatus, not more than 2 mm; upper part of oval window to the utricle (upward), 0.5 mm; posterior part of oval window to the utricle, 1 to 1.5 mm.

For the purpose of comparison, measurements of the stapes footplate may be in order. As seen in Figure 6, the maximum diameter of the oval footplate measures about 2.5 mm to 3 mm and its width about 2 mm. The thickness of the footplate varies considerably; where bone is accumulated near the crurobasal junction, it may be 0.5 mm or more in thickness; where thin (sometimes consisting solely of a plate of cartilage), it may be only 0.0425 mm, or less than 50 micra.

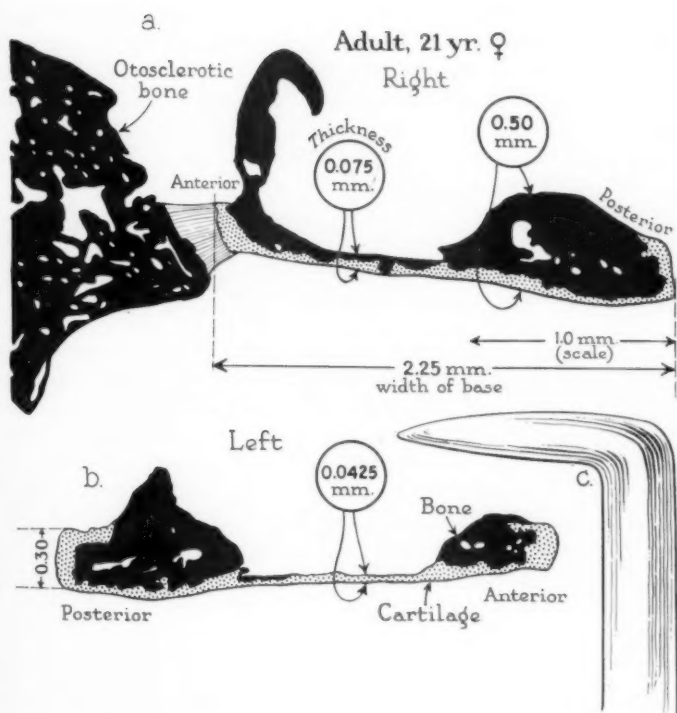


Fig. 6.—Architecture of the footplate of the stapes, concluded, with demonstration of the dimensions of a small fenestrator employed in stapelial mobilization. Adult of 21 years. X 30.

Fig. 6a.—An area in which the base is mainly two-layered. Fig. 6b.—An area (nearer the center of the base) in which cartilage alone is present centrally, both bone and cartilage peripherally. Fig. 6c.—An instrument of smallest dimensions drawn to scale with the stapelial bone.

The thickness of the normal stapedial ligament (i.e., between tympanum and vestibule) is usually about 0.4 mm.

Surgical Significance. The significance of these short distances is not fully appreciated in viewing the middle or internal ear under magnifications such as are used in stapes mobilizations or fenestrations, or in examining such enlargements as are shown in these illustrations (Fig. 6a, 6b). Certainly practice trials would convince the endaural surgeon that manipulating a fine hook or needle or chisel (Fig. 6c) held with the finger some distance from its point, in an attempt to move or pierce a resistant object to a distance of a millimeter or less, will require an unusually accurate kinesthetic sense, or control such as few possess without long training. This is true all the more when the resistance of the object is unknown.

Furthermore, in the event of a break through the stapedial footplate or ligament, the direction and depth of the thrust is of great significance. It would seem that there are two specially vulnerable directions: one is at the anterior part of the oval window where a penetration of 1 mm would reach the sacculus, and a penetration of 2 mm would reach the internal auditory meatus (with its cerebrospinal fluid) and the emerging saccular nerve; the other is at the upper margin of the footplate where an upward thrust is within one-half millimeter of the point of entrance of the utricular nerve into the macula of the utriculus.

SUMMARY

The authors have presented an account of the transformation of the annular cartilaginous stapes of the early embryo into a massive ossifying structure during mid-fetal life and the final conversion, shortly before birth, into the fragile ossicle as seen in the adult. The stapedial base (footplate) of the mature ossicle is a thin oval plate whose greater and lesser diameter measure about 2.75 and 2 mm respectively and whose thickness varies from 0.0425 mm to 0.5 mm.

The total volume of a cast of the bony labyrinth displaces about 0.2 cc or about three drops of water. The actual fluid content is considerably less.

Distances between various parts of the labyrinth are excessively small; as a consequence, probes inserted through the stapes can easily

penetrate vital parts. For example, a probe thrust through the oval window in an anterior direction will enter the saccule within a distance of 1 mm or less and the internal auditory meatus within 2 mm or less. An upward thrust of the probe through the upper part of the stapes will puncture the utricule within a distance of 0.5 mm.

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1. Contribution from the Department of Anatomy, Northwestern University Medical School and the Department of Anatomy, University of Wisconsin (Contribution No. 633 from the former). A study supported by grants from the Central Bureau of Research of the American Otological Society, from the National Institute of Health of the United States Public Health Service and from the Wisconsin Alumni Research Foundation. Art work was executed by Jean McConnell and Rosamond Howland.

The illustrations were prepared from the following series in the Wisconsin Collection: Fig. 1a, series P37; Fig. 1b, ser. P9; Figs. 2a to 2c, ser. P46; Figs. 3, 4, 5a and 5b, ser. 124; Fig. 6a, ser. P28; Fig. 6b, ser. P9.

2. The illustrations upon which this condensed account is based are figures 1a through 3c which appeared in an article by S. F. Richany and the present authors in the Quarterly Bulletin of Northwestern University Medical School, 28:1:17-45, 1954.

XXXII

REPAIR AND CONSEQUENCES OF SURGICAL TRAUMA
TO THE OSSICLES AND OVAL WINDOW
OF EXPERIMENTAL ANIMALS

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In 1824 Flourens⁷ discovered that a pigeon with its columella removed could hear. This observation was made incidentally. At the time of the discovery, the investigator's main objective was to find out why the small birds, on which he was experimenting, reacted so violently to his cutting the VIII nerve. Flourens had been studying the effect of cutting each of the paired spinal nerves from tail to head, and then the intracranial nerves, in turn. Thus he discovered the function of the semicircular canals, and made the observation pertinent to our work.

Kessel,¹⁰ writing in the *Archives für Ohrenheilkunde* in 1876, stated that he had diligently read the works of Flourens, Goltz and von Breuer and as early as 1871 had performed animal experiments on the dog and on pigeons. He had studied the effect of removing the stapes and the columella in these animals. He concluded that after the eighth day a membrane must have formed over the oval window and that the animal heard. He tested the hearing by whistling and creating sounds when the animal was either asleep or sitting quietly. He also used a pistol shot for the test. Apparently he continued the experiments only during one month (June) and does not state what finally became of the animals.

This work was made possible through a research grant of the Public Health Service, U. S. Dept. of Health, Education and Welfare.

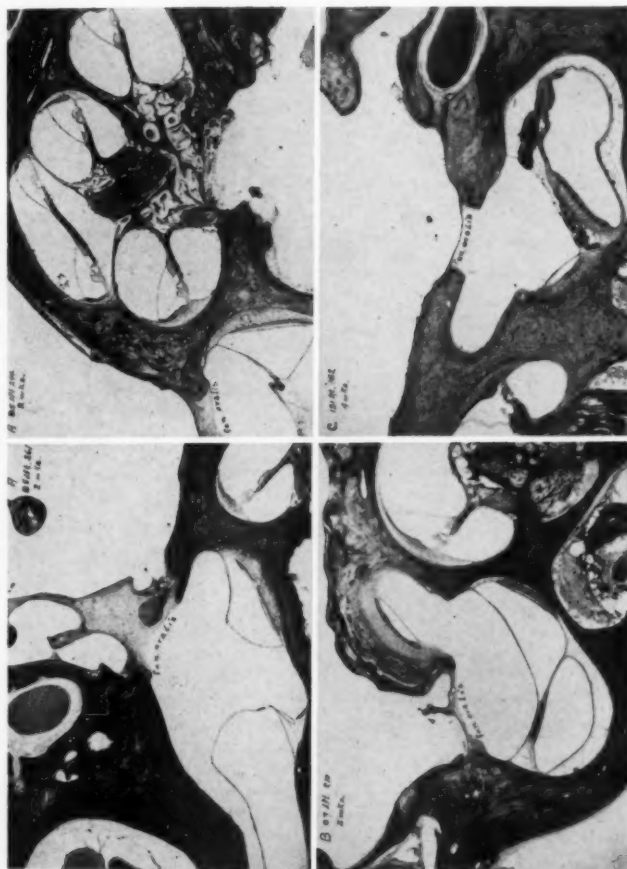


Fig. 1.—Removal of the stapes from oval window is followed by the formation of a membrane across the window. The integrity of the membranous labyrinth is maintained so long as it is not traumatized. The postoperative time in the above cases was 2, 3 and 4 weeks.

Hermann Schwartz in his "Handbuch für Ohrenheilkunde," Volume II, published in 1892, stated on page 785, line 29, that he had first recommended improvement of hearing through "Sondierung," mobilization of the stapes in 1870. He said this possibility had long been recognized. Schwartz referred to Kessel's article entitled "Mobilisierung des Steigbügels" where Kessel described cutting adhesions in the human stapes, separation of the incudostapedial joint and tenotomy or cutting of the stapedial tendon. In a footnote Schwartz referred to a paper published in 1879 by Kessel on the extraction of the stapes in 16 cases of stapedial ankylosis.

Schwartz also referred to work by French surgeons, Bourcheron and Miot,^{3,11} who mobilized the stapes by pressure, using a kind of direct passive movement of the stapes by means of a hook placed between the crura. Actual sclerosis of the stapes was considered a contraindication for mobilization by Miot. In discussing the procedure Schwartz spoke of removal of the drum membrane and frequent consequent suppuration with labyrinthitis and meningitis developing.

In 1939 Engström⁶ reviewed the whole literature of otosclerosis. He illustrated the fractured promontory of an experimental cat but failed to state just what he had done to the animal and did not illustrate the oval window. The illustration does show the fracture line on the promontory and a jagged piece of new bone growing on the inner wall of the inner ear. All of the many other illustrations are from cases of human otosclerosis.

In 1954 Hans Brunner⁴ reviewed the subject of stapedial removal. He illustrated a human microscopic section from his European collection showing a membrane crossing the oval window after stapedectomy.

In 1955 Muerman and Muerman¹² presented a comprehensive historical review of stapes mobilization stating that Miot in 1890 had improved the hearing of 28 cases out of 56 patients operated upon.

In 1956 P. E. Meltzer gave a short historical review of this subject, referring particularly to the Americans, Jack and Clarence Blake.² He also referred to the work of Urbantschitsch and Gelle.

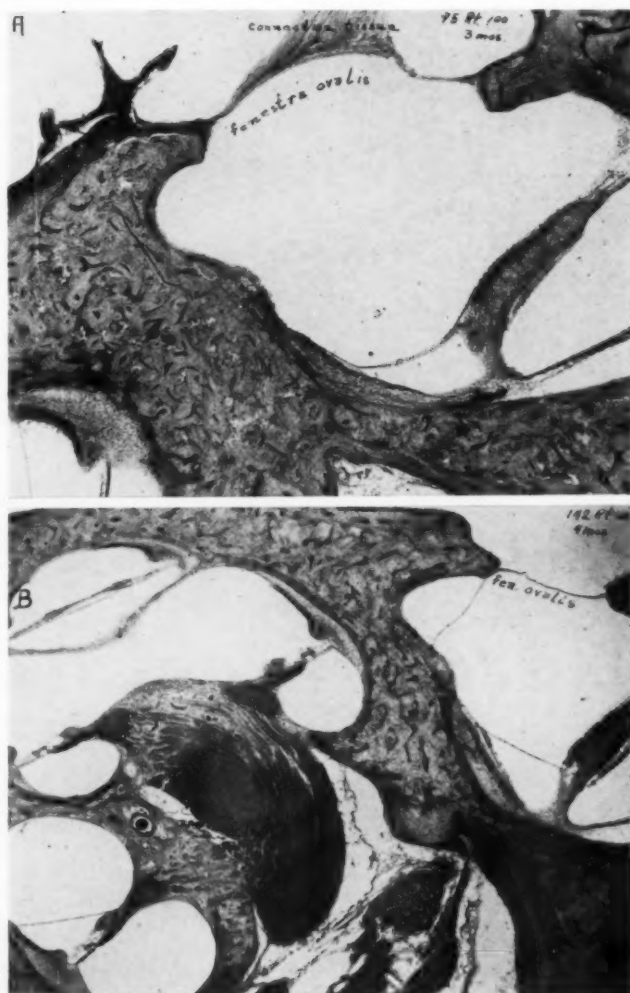


Fig. 2.—The membrane across the oval window as it appears three and four months postoperatively. The membrane is of dual origin. It is formed from submucosa of the middle ear and endosteum of the inner ear.

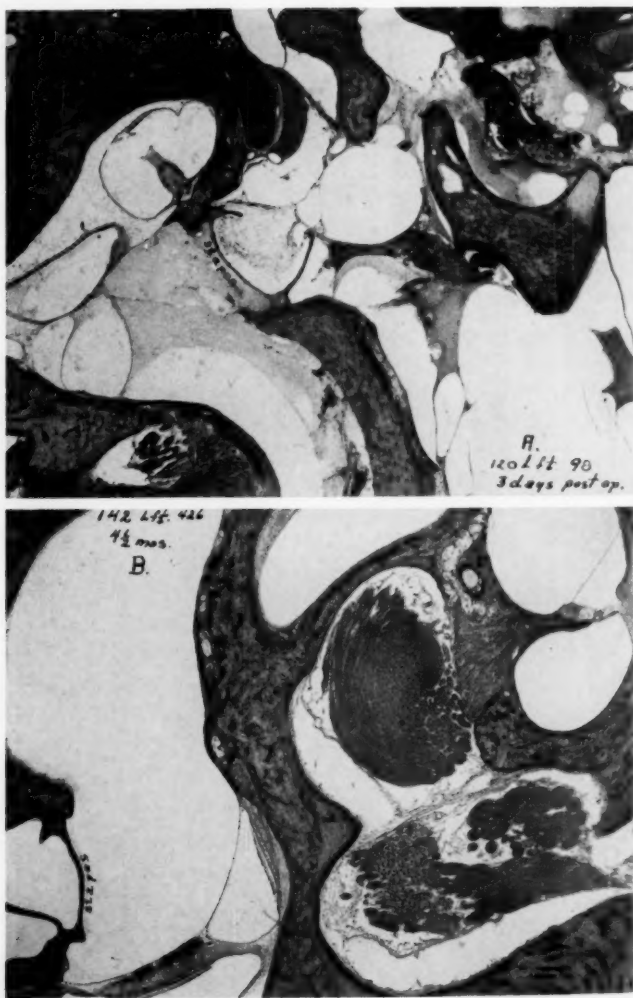


Fig. 3.—Removal of the stapes from the oval window and immediate replacement. Illustration A shows the condition three days postoperatively. Illustration B shows a case 4½ months postoperatively. Ankylosis had developed in the anterior end of the footplate at this site. The rest of the articulation was normal in appearance.

With the exception of the two articles cited above we have found no reports on experimental mobilization on animals. We have found no microscopic illustrations showing the timed results of such experiments.

In 1953 Rosen¹³ reintroduced a mobilization procedure for otosclerosis. Throughout the world mobilization procedures have been followed with varying modifications. Therefore in recent years the labyrinth and the structures within the middle ear have been exposed to various types and degrees of trauma as a result of modern surgical procedures for the restoration of hearing.

In most cases, without too much consideration of the final result of this type of trauma, the crura of the stapes have been fractured, the footplate of the stapes has been cracked and deliberately fenestrated with consequent loss of perilymph. In some cases the stapes has been dislocated or displaced leaving the oval window unprotected and uncovered. In others the ossicular articulations have been separated.

In human surgery, rarely is the surgeon permitted an opportunity to observe healing processes following these injuries. So far as we know, little, if any, experimentation has been undertaken on animals to determine the consequences and subsequent repair of such trauma.

Experimental data is essential in order to continue the present surgical techniques with confidence and to establish some factual basis for further progress in this field of otology. We recognize that results obtained in experimental animals cannot be interpreted as the same as those which might accrue in human surgery. Nevertheless, information is gained when animals are exposed to injuries similar to those which occur in the course of surgery on the human ear. Nature's method of healing and repair and the response of the delicate inner end-organs to trauma can be studied in detail.

EXPERIMENTAL METHODS

The monkey and the cat were selected as suitable laboratory animals as the middle ear can be easily exposed and direct visualization of the ossicles and oval window is possible. Furthermore these animals

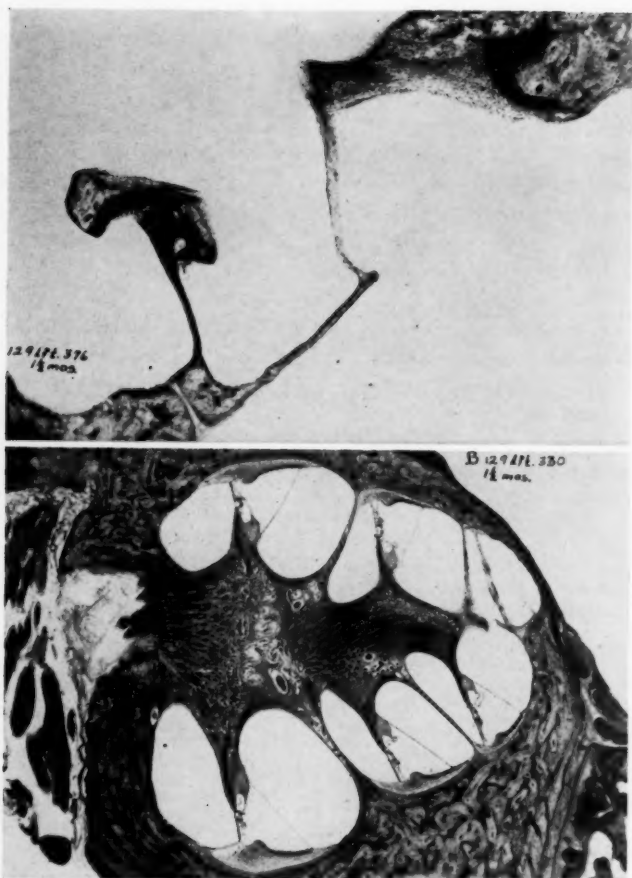


Fig. 4.—When the stapes was fenestrated the footplate was fractured leaving the anterior half of the oval window exposed. This area was covered by a membrane as was observed 1½ months postoperatively. The posterior crus was also fractured. Regeneration of this crus had occurred and new bone was developing on the anterior border of the vestibular wall of the oval window. Cochlea was in good condition with the exception of outer and inner sulcus cells and Hensen's cells in the basal turn.

are responsive and agile, permitting accurate postoperative clinical observations regarding vertigo and hearnig.

All experiments were conducted under sterile operative technique. The animals were anesthetized by intraperitoneal injection of sodium nembutal of appropriate amount. After shaving and preparing the operative area, the middle ear was approached through a postauricular longitudinal incision. The parotid gland was reflected anteriorly and the mastoid bone was entered with a burr at a point just anterior to the emergence of the facial nerve. The incus and stapes were found easily when one-half cm of cortex was removed immediately posterior to the external auditory meatus. The surgical exposure of the stapes was considered very favorable as the oval window niche could be seen through the microscope.

Twenty-four postoperative ears and two control specimens have been included in this series. One of the two monkeys used died on the operating table, immediately following the operation as a result of an overdose of anesthetic. Postoperative observations therefore cover a period from immediately after the operation to eight months following surgery. In several instances movies were taken, the day following the operation and on the day the animal was sacrificed. Occasionally they were taken at intervening periods. Microscopic sections were prepared and examined from all specimens.

Eight different types of operations were performed. In a few instances more than one procedure was carried out on a single ear. The procedure attempted was not always successfully accomplished in every detail. The results in these cases, however, give us valuable information.

The variations in the trauma deliberately induced on the middle ear structures are listed as follows:

A. Removal of the stapes from the oval window (Figs. 1 and 2).

B. Removal of the stapes from the oval window and immediate replacement of it (Fig. 3).

C. Removal of the stapes, "fenestration" (hole) made in the footplate and replacement of the stapes (Fig. 4).

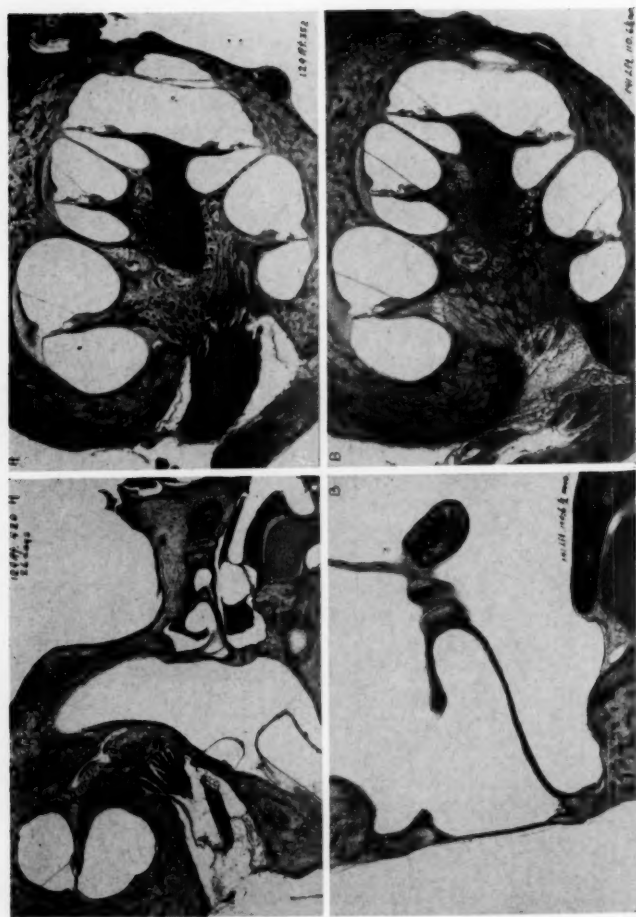


Fig. 5.—Fracture of the anterior crus. Disarticulation and realignment of the incudostapedial joint. Post mortem microscopic examination shows good alignment in both cases. Note the connective tissue in which the specimen of 26 days is embedded (A)

OBSERVATIONS:

A.

REMOVAL OF STAPES FROM OVAL WINDOW

NO. OF SPECIMEN	ADDITIVE POSTOPER- ATIVE TIME	RESULTS	
1. 87 Rt. Mky. #2	0	No stapes in situ. Normal middle and inner ear. Hemorrhage into vestibule, especially near cochlea. Reisner's membrane sags slightly over axial part of limbus. Organ of Corti in excellent condition.	
2. 85 Lft. Mky. #1	2 wks.	No stapes in situ. No otitis media. No labyrinthitis. A healthy thick membrane which is connected with a polyp-like growth of uninfected connective tissue in middle ear, crosses the oval window. Organ of Corti excellent throughout. Stray rbc's in cochlea probably entered at autopsy. Slight hemorrhage below posterior crista.	
3. 87 Lft. Mky. #2	3 wks.	No stapes in situ. No otitis media. No labyrinthitis. A fibrous band of tissue somewhat columella-shaped extends across the oval window. End-organs of the inner ear in good condition. Protoplasm of the inner hair cell appears reduced in amount. Inner sulcus cells are deficient. Spiral ganglion normal.	
4. 131 Rt. Cat #10	1 mo. 1 wk.	No stapes in situ. No otitis media. No labyrinthitis. Sections 160-188 show a delicate membrane crossing the oval window. Organ of Corti is in good condition except for dislodgement of inner sulcus cells.	
5. 112 Lft. Cat #6	1 mo. 3 wks.	No stapes in situ. No otitis media. No labyrinthitis. A very delicate membrane, apparently derived primarily from endosteum, and excessive in length, covers the oval window at the lower level. At a higher level a thicker membrane derived from both mucosa and endosteum is present. Organ of Corti is damaged in all coils	

A. (Continued)

NO. OF SPECIMEN	POSTOP. TIME	RESULTS
6. 95 Rt. Cat #1	3 mos.	No stapes in situ. No otitis media. No labyrinthitis. A rather sturdy membrane, columella-shaped crosses the oval window. Organ of Corti is in fair condition throughout. Vestibular membrane sags over tectorium. Outer wall of sacculus is depressed and there is some atrophy of the macula.
7. #142 Rt. Cat #9	4 mos.	No stapes in situ. No otitis media. No labyrinthitis. A delicate membrane has formed across the oval window. It is slightly longer than the distance required. Organ of Corti is in fair condition except in basal turn where Hensen's cells are completely dislodged. Tectorium is missing or in a state of dissolution, higher up or more apicalward in the cochlea. Reissner's membrane is in situ although there is evidence of edema of perilymphatic spaces in vestibule.
8. #141 Rt. Cat #5	8 mos.	No stapes in situ. No otitis media. No labyrinthitis. Oval window covered by a very delicate membrane which in places extends up around the facial canal. Organ of Corti shows normal inner hair cell, normal pillars, low fused Hensen's cells and fused outer hair cells. No tectorium can be seen. Technically this specimen was incompletely infiltrated. It was possible to see, however, that the organ of Corti was of normal height. Its cells could not be clearly differentiated.

B.

STAPES REMOVED AND IMMEDIATELY REPLACED

NO. OF SPECIMEN	ADDITIVE POSTOPER- ATIVE TIME	RESULTS
1. #120 Lft. Cat #8	3 days	Fairly accurately replaced except for one slight area. The part of the oval window not completely covered by the replaced footplate was covered by a delicate membrane. (This is important since the postoperative time was only three days.) Organ of Corti shows post mortem degeneration in the operated and unoperated side. Found dead Monday morning. Animal in good condition Friday previous.
2. #142 Lft. Cat #9	4 1/2 mos.	Stapes perfectly aligned posteriorly. Signs of early ankylosis at one level, anteriorly. More inferiorly it appears normal. Organ of Corti in basal coil shows marked edema of outer and inner sulcus cells and Hensen's cells. More normal at higher levels.

C.

STAPES REMOVED AND FOOTPLATE "FENESTRATED"

1. #129 Lft. Cat #11	1 1/2 mos.	Anterior half of stapes is completely missing from oval window. Posterior crus and posterior half of footplate are in situ. A delicate membrane covers the part of oval window where no footplate lies. The existing part of the footplate is fractured in three places. There is evidence of deposition of new bone by accretion at the broken end of the footplate and over some of the fractured areas; also at the distal end of the fractured crus. New bone is appearing on the anterior vestibular wall.
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D.

ANTERIOR STAPEDIAL CRUS FRACTURED BUT FRAGMENTS NOT DISPLACED.
INCUDOSTAPEDIAL JOINT DISARTICULATED AND REALIGNED

NO. OF SPECIMEN	POSTOP. TIME	RESULTS
1. # 129 Rt. Cat # 11	26 days	<p>The fragments of the anterior crus are suspended in connective tissue. New bone by accretion has been deposited at the inner base of the crus (386). The posterior crus is also embedded in connective tissue as are the parts of the incudostapedial articulation which are perfectly aligned. There is no evidence of infection in middle or inner ear. Organ of Corti is in fair condition throughout and M. vestibularis is in situ. In the basal coil, however, Hensen's cells are heaped upward as if dragged in this position by tectorium which is disintegrated. Tectorium is also detached from limbus in apical coil but in situ in intervening areas. An unhealed fracture is present in the middle of stapedial footplate.</p> <p>is present in the middle of stapedial footplate. Two similar fractures are on the opposite stapedial footplate which, however, was fenestrated in the footplate.</p>
2. # 141 Lft. Cat # 5	6½ mos.	<p>Incudostapedial joint perfectly aligned. No longer embedded in connective tissue. Anterior crus as seen in section 150 shows fragments of old bone of crus embedded in new bone deposited by accretion. Organ of Corti in fair condition throughout but outer and inner sulcus cells edematous. Tectorium missing in basal turn. Plasma coagulated between outermost outer hair cell and next one. Stria fails to exhibit normal sections of vascular channels. There is marked edema of perilymphatic trabeculae in the vestibular labyrinth (312-410).</p>

E.

FRACTURE OF BOTH STAPEDIAL CRURA
BETWEEN THE BASAL ONE-THIRD AND DISTAL TWO-THIRDS

NO. OF SPECIMEN	POSTOP. TIME	RESULTS
1. #139 Lft. Cat #7	6 mos.	<p>No pus in the middle ear but there is evidence of a chronic lymphoid infiltration in the mucosa of the eustachian tube and outer bulla. This is not related to the operative procedure. It is systemic. No labyrinthitis is present.</p> <p>A fenestration of the horizontal canal was inadvertently made while approaching the oval window. This fenestra is now almost completely closed (sections 199-210-218). Facial nerve is completely denuded and damaged in one region.</p> <p>Anterior crus of stapes is fractured near the base (210). Posterior crus is fractured at this level and at a slightly higher level (218). On the anterior crus new bone has been deposited by accretion and can be clearly differentiated from the mature bone. The new bone has formed across the stapedia vestibular joint and has firmly ankylosed this joint.</p> <p>Organ of Corti is of normal height throughout except that Hensen's cells in the basal turn are reduced and fused with the two outer hair cells. External sulcus cells are edematous and sloughed off. Stria vascularis shows signs of sloughing and this condition grows progressively worse toward the apex. Reissner's membrane is in situ throughout and tectorium is in fair position.</p>

F.

FRACTURE OF POSTERIOR STAPEDIAL CRUS

NO. OF SPECIMEN	POSTOP. TIME	RESULTS
1. #139 Rt. Cat #7	7 mos.	<p>Otitis media is present with cholesterolin crystal imprints between the stapedial crura and throughout the niche of oval window. There is a voluminous lymphadenitis in this middle ear with well organized lymph nodules present. Outer bulla is completely filled with scar tissue. No Langhaus giant cells are identified but indications are that this cat had a systemic lymphadenitis.</p> <p>There is no purulent labyrinthitis. A slight granular precipitate is present in the cisterna perilymphatica. Organe of Corti is in fair condition throughout. The basal turn shows the same degeneration of Hensen's cells in basal coil and of stria as on the opposite ear.</p> <p>As shown in section 181, 186 and 189 the anterior crus has been fractured. New bone has been deposited by accretion on the posterior side of the crus, not on the anterior surface and no ankylosis has occurred. Incudostapedial joint is in perfect alignment (190). Posterior crus is less fractured than anterior, being merely cracked and the posterior part of the footplate has been slightly cracked. The posterior vestibular wall has also been fractured slightly (181). Early ankylosis is indicated, however, at the posterior stapediostapedial joint in this section.</p>

G.
PRODUCTION OF TRAUMA TO BONE IN THE NICHE OF OVAL WINDOW

NO. OF SPECIMEN	ADDITIVE		TRAUMA INDUCED	RESULTS
	POSTOP. TIME			
1. 95 Lft. Cat #1	3 wks. 5 da.		Crura fractured. Wall of oval window roughened.	<p>Promontory has been fractured in two places and a piece of bone thrust into the spiral ligament. Over the oval window there are numerous fragments of bone embedded in dense fibrous tissue. Around the fragments new bone is being deposited by accretion.</p> <p>In the cochlea Reissner's membrane is distended in the middle coil so that it touches the apical surface of scala vestibuli. Neither outer nor inner hair cells are present. Hensen's cells are present and maintain the normal contour of organ of Corti. There are free blood cells in the scala vestibuli and scala tympani.</p>

NO. OF SPECIMEN	ADDITIVE		TRAUMA EXERTED	RESULTS
	POSTOP. TIME			
2. 131 Lft. Cat #10	7 wks.		Footplate of stapes left intact. Stapediovestibular articulation traumatized from mid-position of anterior superior aspect to midposition of anterior inferior aspect.	<p>Footplate was inadvertently fractured and one crus thrust within the vestibule (172-184). It rests upon but does not penetrate the outer wall of sacculus. The vestibular wall was fenestrated superiorly just beneath the macula of utricle (160-162). Dense fibrous tissue covers the oval window and within this in the vestibule lies the anterior crus and an extensive part of the footplate of the stapes. Section 196 shows that new bone had crossed the niche of oval window lateral to the proper location of stapedial footplate. The footplate is in situ at this level.</p> <p>In the cochlea Reissner's membrane sags over limbus in all coils. Outer and inner sulcus cells are edematous and necrotic. Hensen's cells and sustentacular cells (Deiter's) are also highly edematous and necrotic. Outer and inner hair cells, however, are easily identified (194).</p>

H.

STAPES THRUST INTO VESTIBULE THROUGH OVAL WINDOW

NO. OF SPECIMEN	ADDITIVE POSTOP. TIME	RESULTS
1. 85 Rt. Mky. #1	3 wks.	No otitis media. No labyrinthitis. Footplate was shoved deep within vestibule along with many fragments of bone. The lumen of the sacculle was invaded as well as the anterior part of cisterna. New bone was being deposited by accretion around these fragments of bone. Organ of Corti appears excessively high due to edema of Hensen's cells. Nuel's space is obliterated. Outer sulcus cells are edematous. Tectorium appears to be undergoing liquefaction and the tier of cells in the limbus are hyperactive. Reissner's membrane is in situ.
2. 88 Rt. Cat #2	6 wks.	No otitis media. No labyrinthitis. Footplate has been shoved into cisterna and touches the base of but does not damage macula utriculi. This cat's horizontal canal was also inadvertently fenestrated (268) and the facial nerve was slightly damaged. As in the previous case Nuel's space is obliterated. Hensen's cells are edematous as are outer sulcus cells. Deiter's cells cannot be identified although the nuclei of the three outer hair cells can be seen. Tectorium is undergoing liquefaction and many of the cells in limbus are necrotic. Reissner's membrane is in situ.
3. 131 Lft. Cat #10	7 wks.	No otitis media. No labyrinthitis. Round cell infiltration in submucosa of middle ear. Anterior crus and footplate lie embedded in connective tissue across denuded part of oval window. They are embedded in dense connective tissue. The footplate impinges slightly on saccular wall but does not rupture it. Organ of Corti in fair condition throughout. Outer and inner sulcus cells and Hensen's cells are edematous in basal turn. Reissner's membrane sags over limbus and tectorium. Tectorium in basal coil appears normal. At higher levels it is edematous. The sustentacular cells of the hair cells (outer and inner) are missing. There is no Nuel's space.



Fig. 6.—Both stapedial crura were fractured between basal one-third and distal two-thirds. The crura had both begun to regenerate six months later. New bone was deposited by accretion and in this process ankylosis of the anterior crus of stapes developed.



Fig. 7.—Posterior stapedia crus was fractured. Actually the anterior crus was also fractured and its component fragments were displaced. Regeneration of this crus had begun. It proceeded by accretion on the posterior wall of the anterior crus; therefore no ankylosis occurred. The two cracks in the posterior crus had not healed. Note otitis media.

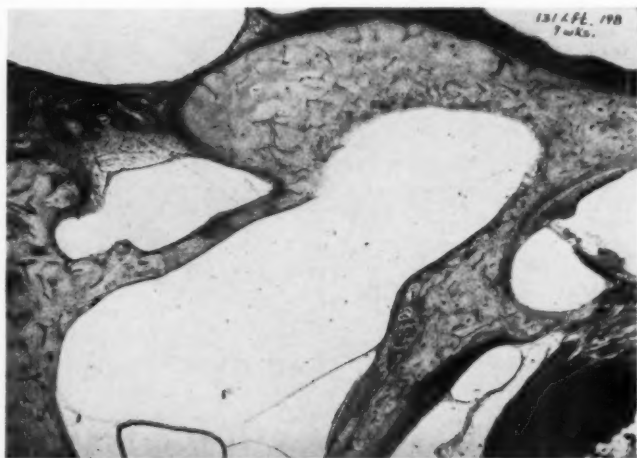


Fig. 8.—Recently formed bone lies lateral to the footplate of the stapes, seven weeks after the edges of the niche had been roughened to determine if trauma in this region predisposed to growth of new bone.

D. Fracture of anterior crus of the stapes. No displacement of fragments. Disarticulation and realignment of incudostapedial joint (Fig. 5).

E. Fracture of both stapedial crura between basal one-third and distal two-thirds (Fig. 6).

F. Fracture of posterior stapedial crus. (Inadvertently fractured anterior crus at higher level than the posterior) (Fig. 7).

G. Production of trauma to bone in niche of oval window (Fig. 8).

H. Depression of stapes into vestibule (Fig. 9).

COMMENT

Proper evaluation of results from the procedures carried out in these experiments necessitated the study of complete series of sections through each petrosa. Just as in the evaluation of the extent of a

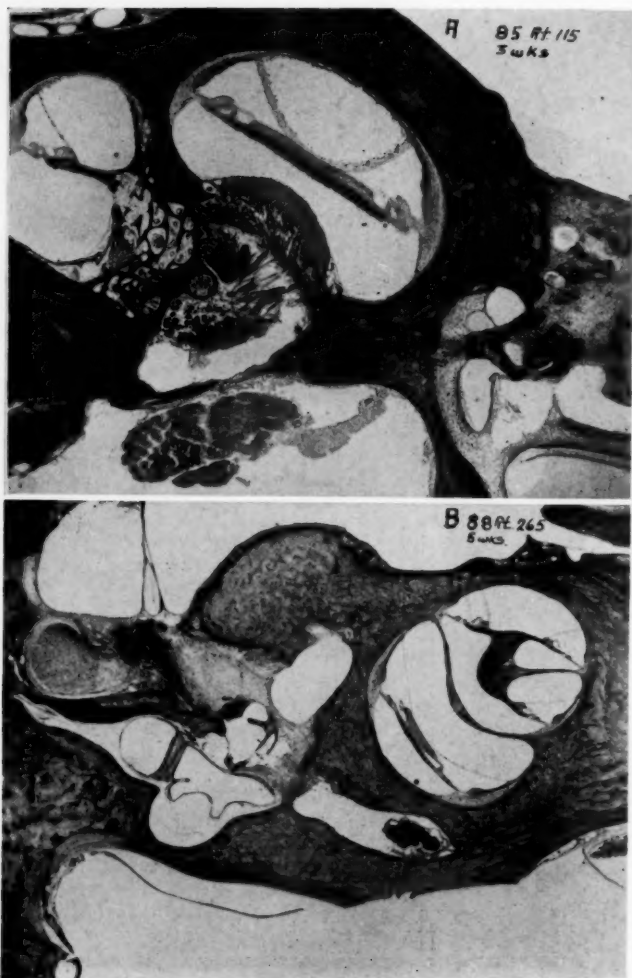


Fig. 9.—The stapes becomes embedded in dense fibrous tissue when thrust within the oval window. If damage is perpetrated upon the membranous labyrinth, particularly upon the saccule as in A, this damage is reflected in the cochlea and organ of Corti. The postoperative time in A was three weeks. The postoperative time in B was five weeks.

lesion of otosclerosis in post mortem examination, so also in these microscopic observations, it was necessary to examine all levels of the membranous labyrinth (Fig. 11).

The motion pictures gave further confirmation as to the ability of the animals to adjust to postoperative conditions. Our experiments did not include measurements of electrical cochlear responses to sound. The animals became laboratory pets, however, and definitely responded to unmeasured sound stimuli.

In these experiments it was evident that when a stapes was carefully removed from the oval window and the perilymph was not disturbed by suction or manipulation, no appreciable amount of fluid was observed to escape. Reissner's membrane remained in situ, the membranous labyrinth retained its normal shape and the end-organs appeared to be normal (Fig. 12).

Brunner in speaking of stapedectomy of the human ear stated that "where the stapes comes away intact it is lifted from its attachment with a sense of suction resistance." Blake observed "perilymph" drain for ten days from the oval window of one of his human cases.

Our experiments reveal that a prompt response to trauma in the middle ear was the formation of dense connective tissue. This tissue was found principally in the traumatized area and was observed to fill in the niche of oval window (Figs. 5A, 9, 10B and 11A). It was later resorbed and by the fourth week the middle ear cavity had usually returned to the normal state (Figs. 1C, 2B and 5B). The oval window after stapedectomy developed a protective membrane as early as three days postoperatively. This was in the area not occupied by the replaced stapes of 3A (see Figure 12 also). The original mass of connective tissue which filled the niche of the oval window was observed to act as a medium for the more permanent membrane. This structure was of dual origin, derived partially from the middle ear submucosa and partially from the endosteum which formed the inner layer (Fig. 2B). With time the membrane was observed to undergo a process of thinning. All denuded oval windows (16 in number) were covered by the new membrane with the exception of one infected ear. In this specimen a fragment of the stapes was observed to lie within the vestibule (Fig. 11). It is worthy of note that no labyrinthitis is evident in the cochlea two weeks after the operation.

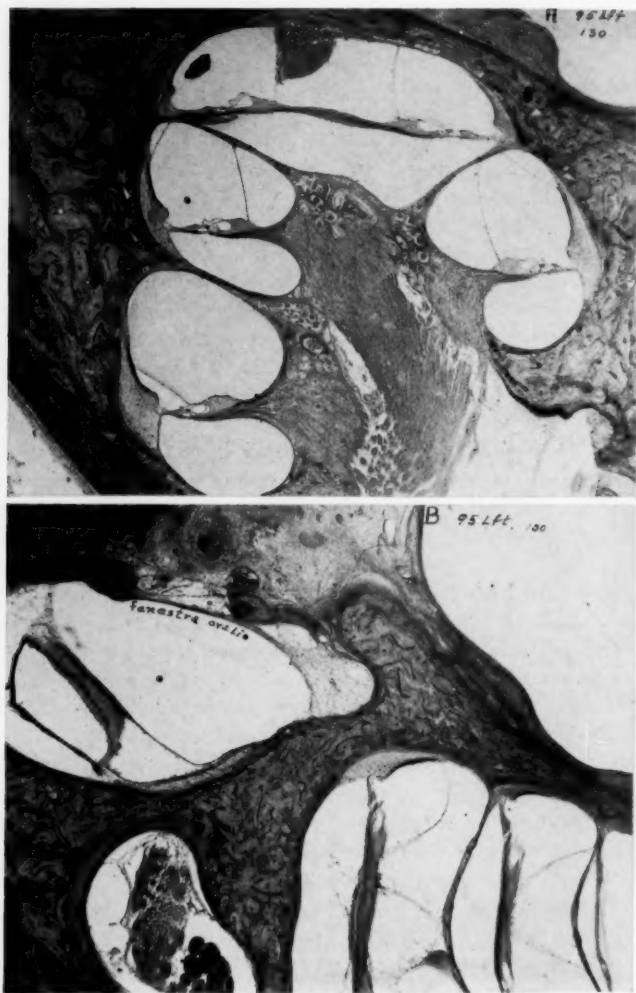


Fig. 10.—Stapedial crura were fractured and the walls of the niche of oval window were roughened. In so doing the promontory was fractured in two places and fragments of bone thrust into spiral ligament and saccule. Note the state of Reissner's membrane. The cat was normally active 25 days postoperative but had shown vertical nystagmus earlier.

When the stapes was removed it was difficult to replace it accurately (Figs. 3 and 12). If an uncovered space remained between the edge of the footplate and the vestibular rim, this space was promptly closed by a delicate membrane. In one case the footplate of the stapes made contact with the vestibular wall and early ankylosis was noted at this site (Fig. 3A). The rest of the stapediovestibular joint presented a normal appearance.

Fracture of the stapes was usually followed by accretion of new bone (Figs. 4, 6A and 7B). This repair occurred whether the fracture was at the footplate or in the crura. In one case where the anterior crus was fractured repair took place by accretion of new bone (Fig. 7B). As the fracture was near the footplate this repair process involved the vestibular wall causing ankylosis of the stapes (Fig. 6A). In another case, when the posterior crus was experimentally fractured, two cracks of this crus were observed in the post mortem microscopic sections. The anterior crus of this same stapes, however, was completely broken off and the component parts separated. In the process of healing, new bone was deposited by accretion on the posterior surface of the anterior crus but in this case, however, no ankylosis developed. Two fractures were observed which had not healed 26 days postoperatively and two, seven months postoperatively.

The delicate membranous labyrinth and the end-organs presented more resistance to trauma and infection than was anticipated. Sterile surgery yielded good results. In the course of our experiment purulent labyrinthitis was found in only two ears. In one of these cases infection developed when mucosa from the mouth was placed over the oval window. In the other case, the labyrinthitis was so mild that we did not realize it had developed. In this case nystagmus was not observed. Meningitis did not develop in any of the experimental animals. Otitis media developed in four ears following stapedectomy but no labyrinthitis was observed. Animals which developed middle ear infection were observed over a period from five days to three weeks postoperatively before being sacrificed.

On occasions when inner ear organs were traumatized it was the saccule rather than the utricle or ampulla of the lateral canal which was more frequently injured. If the wall of the saccule was compressed by the presence of a stapes depressed into the vestibule or when displaced by bone fragments, resting on the spiral ligament at

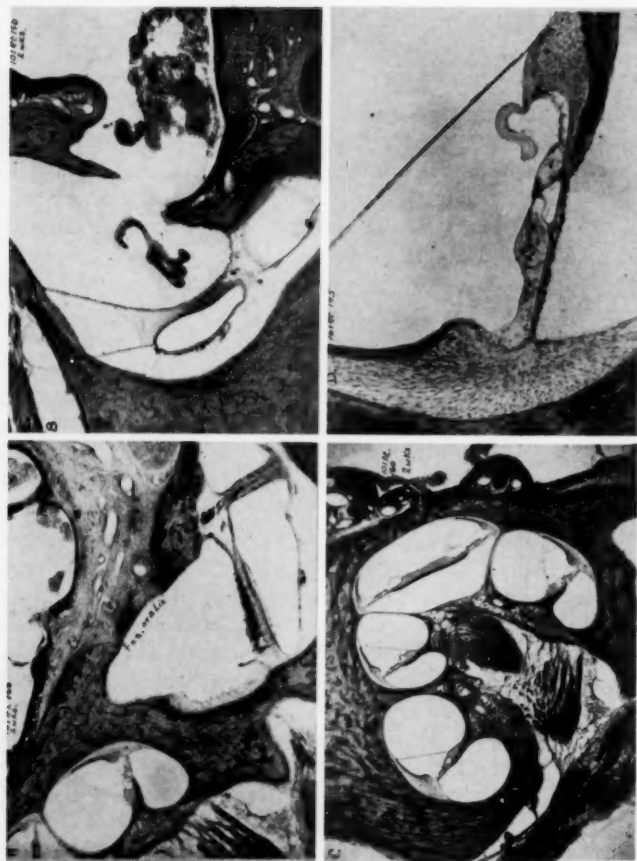


Fig. 11.—The necessity for study of all levels of the labyrinth is evident from this specimen. In A at the level of section 160 the membrane covers the oval window but is entirely absent of stapes media. In B the membrane is absent and a fragment of stapes lies within the vestibule. In C the nuclei of inner hair cells are visible. In D the innermost three outer hair cells are visible. The nuclei of inner hair cells is necrotic however, and the innermost of three outer hair cells is absent.

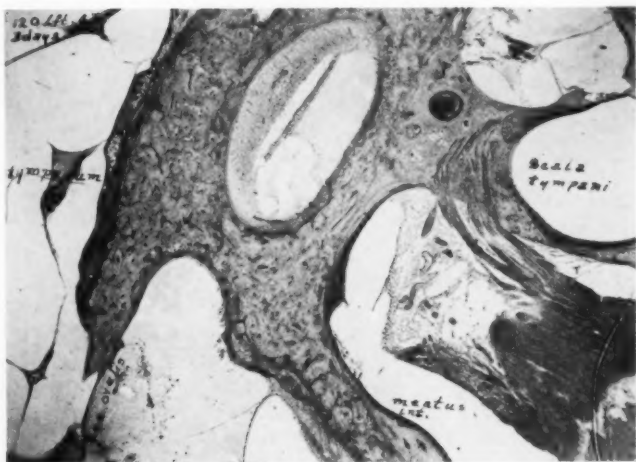


Fig. 12.—In the lower left corner of the illustration a thin membrane covers the oval window. The case is also illustrated in Fig. 3A where the stapes is seen to be imperfectly placed in the window. The membrane shown in Figure 12 filled in the uncovered part of the window, three days postoperative. Note the distention of Reissner's membrane in basal coil.

the basal turn of the cochlea, then pressure was exerted on Reissner's membrane from within and it was observed to be very greatly distended (Fig. 10). In these few cases the organ of Corti also showed damage, particularly in the basal turn. The structure of the tectorium was affected by these procedures and the appearance of the tiers of nuclei in the limbus was also altered.

The European literature has long maintained that a fractured footplate does not heal. In 1954 Brunner stated that both the extraction and fracture of the stapes do not "invite" the formation of new bone. In 1942 one of us (D. Wolff) reported a case of a riveter who suffered bilateral fracture of the stapes. Post mortem examination revealed that one stapedial footplate had formed a callus. The other stapes was found to have a fractured crus just above the footplate in which no evidence of healing was detected. There was, of course, no means of knowing how long the fractures had been present.

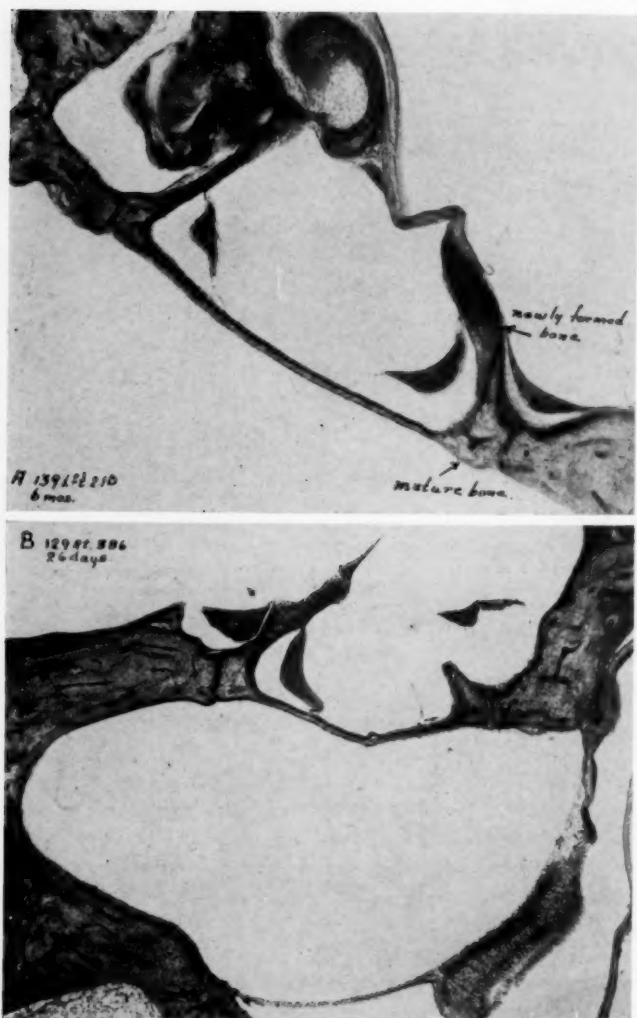


Fig. 13.—Ankylosis of the anterior stapediovestibular joint has occurred in A (139 Lft. 210) 6 months postoperatively. New bone has been deposited by accretion on the fractured crus and this extends across the lateral side of the articulation. Healing has not occurred in the fractured footplate in B (129 Rt. 386) 26 days postoperatively.

The results in our experiments give ample evidence that fractures of the stapes induce a tissue response with the formation of new bone when the fragments are left in situ. New bone was deposited by accretion on the fractured tip of the footplate, following incomplete removal of the stapes. Deposits of new bone and ankylosis were observed following trauma of the vestibular wall at the stapediostapedial joint (Figs. 6A, 8, 10B and 13A).

SUMMARY

1. When the stapes was removed from the oval window only a small quantity of perilymph escaped, if no suction was applied and no agitation was created.

2. Following stapedectomy a membrane formed promptly across the denuded oval window. It was present as early as the third day.

3. When the stapes was removed and replaced ankylosis was observed at the point of contact of the footplate and vestibular wall.

4. When either the footplate or the stapedial crura were fractured, healing in most cases occurred by accretion of new bone; some fractures remained unhealed.

5. When the incudostapedial joint was disarticulated and realigned the ossicles appeared to be in good alignment and the possibility of satisfactory function was good.

6. Trauma to the walls of the niche of the oval window led to the formation of bony bridges in the niche. Fragments of bone displaced into the labyrinth showed evidence of growth.

7. Depression of the stapes into the vestibule induced damage in the inner ear when the vestibular membranous labyrinth was traumatized. When not directly traumatized in such manner, the end-organs exhibited a remarkable resistance to manipulation of the stapes and oval window.

CONCLUSIONS

1. Trauma to the stapes and the oval window is followed by an active repair process which begins promptly and apparently protects

the labyrinth from excessive damage. Vestibular and cochlear damage, however, does not occur when these organs are traumatized directly by instrumentation and by the displacement of bony fragments.

2. Trauma to the stapes and oval window was found to induce the formation of new bone by accretion and subsequent ankylosis occurred.

210 EAST 64TH ST.

REFERENCES

1. Bellucci, R.: Present Status of the Operation for Mobilization of the Stapes. *Laryng.* 66:269-292, 1956.
2. Blake, C. J.: Operation for Removal of Stapes. *Trans. Am. Otol. Soc. Boston Med. and Surg. J.* 127:469,551, 1892. - Middle Ear Operations. *Trans. Am. Ophth. and Otol. Soc.* 5:306, 1891-92. - Stapedectomy and Other Middle Ear Operations. *Trans. Am. Otol. Soc.* 5:464, 1893.
3. Bourcheron: La mobilisation de l'étrier. *Bull. Med.* 2:1225, 1888.
4. Brunner, Hans: Attachment of the Stapes to the Oval Window. *Arch. Otolaryng.* 59:18-29, 1954.
5. Burnett, C. H.: Partial Myringotomy and Removal of the Incus and Stapes for Relief of Lesions of Chronic Catarrhal Otitis Media. *M. News* 62:500, 1893.
6. Engström, H.: Über des Vorkommen der Otosklerosis nebst exper. Studien über Chirur-gachsbehandlung der Krankheit. *Acta Oto-laryng. Suppl.* 43, 1940.
7. Flourens, P.: Recherchés sur les proprietes et les fonctions du system nerveux. Recherchés sur les conditions fondamentals de l'audition, Ch. 17, 1842. *Memoirs de l'academie des sciences de l'inst. de France, Paris*, Vol. 9, p. 5, 1828.
8. Fowler, E. P.: Anterior Crurotomy and Mobilization of the Stapes. *Acta Oto-laryng.* 46:319-322, 1956.
9. Jack, F. L.: Removal of the Stapes in Chronic Otitis Media and Chronic Suppurative Otitis Media. *Boston Med. and Surg. J.*, 1892. - Remarkable Improvement in Hearing by Removal of Stapes. *Trans. Am. Otol. Soc.*, 1891-2, also 1893, 1894.
10. Kessel, J.: Ueber die Durchschneidung des Steigbügelmuskels beim Menschen. *Arch. Ohrenheilk.* 8:231, 1874; 11:199, 1876; and 12:237, 1877.
11. Miot, C.: De la mobilisation de l'etrier. *Rev. de Laryngologie d'otologie* 10, 49, 83, 113, 145 and 200, 1890.
12. Meurman, Y., and Meurman, O.: Stapes Mobilization in Otosclerosis. *Arch. Otolaryng.* 62:164, 1955.

13. Rosen, S.: Mobilization of the Stapes to Restore Hearing in Otosclerosis. N. Y. State J. Med. 53:2650-2653, 1953. - Bergman Restoration of Hearing, etc. An Analysis of Results. Laryng. 65:224-258, 1955.
14. Rosen, S., and Bergman, M.: Mobilization of the Stapes for Otosclerotic Deafness. Acta Oto-laryng. Suppl. 118, 1954.
15. Rosen, S., and Bergman, M.: Improved Hearing after Mobilization of the Stapes in Otosclerotic Deafness. J. Laryngol. and Otology 69:297-308, 1955. - New Middle Ear Mechanisms for Normal Hearing. A.M.A. Arch. of Otolaryng. Vol. 67 (Apr.) 1958.
16. Richards, G. L.: Halle and the Aural Clinic of Professor Schwartze. Boston M. and S. J. 132:273, 1895.
17. Scheer, A. A.: Restoration of Hearing in Otosclerosis by Trans-Tympanic Mobilization of Stapes. Arch. of Otolaryng. 61:513, 1955. - Evaluation of Trans-Tympanic Mobilization of Stapes as Treatment for Otosclerosis. N. Y. State J. of Med. 55:18:2609, 1955.
18. Wolff, D.: Microscopic Examination of the Human Labyrinths from Patients Exposed to Loud Noises. Arch. Otolaryng. 36:843, 1942.

XXXIII

FURTHER STUDIES ON THE FUNCTION
OF THE UTRICULAR MACULA

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Lesions of the nonauditory vestibular organ in the head are practically always discussed as involving some malfunction of the semicircular canals or their central nervous connections. The main reason for this correlation has been due to the fact that clinical tests designed to aid in the diagnosis of vestibular disease have been almost exclusively concerned with determinations of the characteristics of either artificially induced or spontaneous originating nystagmus. The significance of this last statement is based upon the widespread opinion that labyrinthine nystagmus is always induced by semicircular canal stimulation, although Cawthorne and Hallpike (1957) have indicated otherwise. Regardless of this controversy, it has been amply demonstrated that determinations of the characteristics of a patient's reflex eye movements in response to vestibular stimulation constitute the most direct objective means of diagnosis and, as a result, nystagmography has been receiving great prominence of late. However, measurement of eye deviation as distinct from nystagmus in response to vestibular stimulation has been almost completely overlooked in spite of the fact that some evidence exists of its significance as a means of indicating activity of the utricular and possibly saccular maculae.⁶⁻⁸ It is the object of this paper to describe such a procedure and to stress its importance in our understanding of the normal and abnormal activity of the human otoliths.

PROCEDURE

By suspending a newly developed highly sensitive photo-electric element in front of the eye, any ocular movement can be detected

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due to variation in intensity of the light reflected off the anterior surface of the eyeball. The element is so sensitive that critical positional of the device is not required, and since the anterior surface of the eyeball varies in detail from point to point, eye movement causes variations in intensity of reflected light sufficient to activate the photo-sensitive detector, the electrical output of which is fed through an electronic integrator and recorded by a single channel oscillograph.

As seen in Figure 1, a head-piece consisting of an adjustable band provides a firm support for the detecting device on the patient's head. From this band is mounted a light source and photo-cell unit mounted so as to give complete freedom of adjustment on all patients. The light source and photo-cell unit are mounted closely together. The former is positioned to illuminate the eye. The photo-cell is adjusted so that its sensitive region is centered roughly on the margin between the sclera and iris. Horizontal or vertical movements of the eye will thus markedly alter the light/dark ratio as seen by the photo-cell and so produce an electrical output signal from the unit. However, as mentioned above, the unit is so sensitive that critical positioning is not essential, and lack of homogeneity of the anterior surface of the eyeball is sufficient to cause ripples of reflected light sufficient to be readily detectable as the result of any ocular movement.

A small metal case contains the operating controls (Fig. 2), and a stabilized supply of current for the lamp and photo-cell. The power required is the standard 115 volts 60 cycle current. A sensitivity control provides adjustment of the proportion of output which is fed to the recorder. The Reference Adjustment or DC balance control allows balancing of the DC output of the instrument for any given position of the eye. An AC-DC switch allows selection of two modes of operation, as follows: with the switch in the DC position, the output of the instrument is a direct function of the eye position in the selected plane. If it is desired to record small, quick motions of the eye, such as those of nystagmus, in the presence of large changes in the mean position of the eye, the AC position of the switch should be used. This introduces resistance-capacity coupling into the output circuit, thus suppressing the DC component. The values chosen to enable this are such as to provide an output proportioned to the rate of change of the eye position rather than eye position itself. This provides for the maximum reflection of signals due to gross change in the eye position.



Figure 1

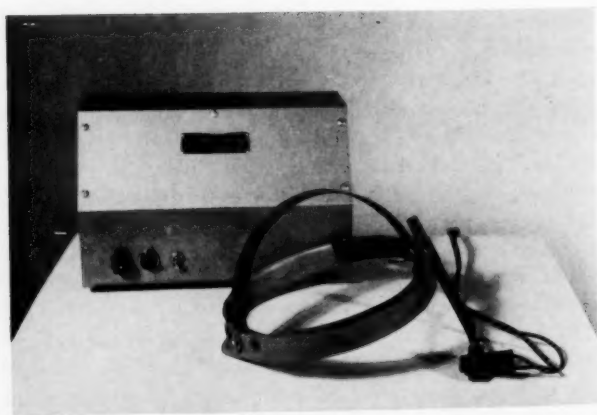


Figure 2

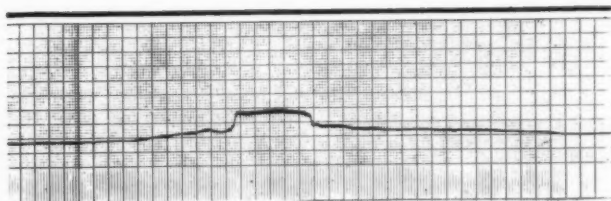


FIGURE 3

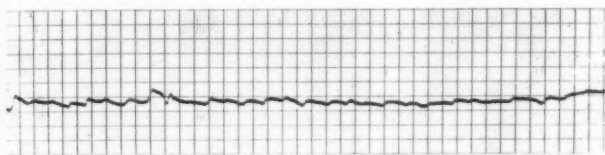


FIGURE 4

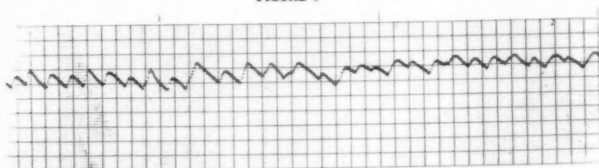


FIGURE 5

The circuit arrangement is such that various types of recorders may be used, although we usually use a direct writing electrocardiogram for this purpose.

The sensitivity of the unit will vary with the optical characteristics of the eye, and to some slight extent with the level of ambient light, but it is normally more than 10 millivolts per degree of eye movement when the unit is connected to a one-megohm load which approximates the characteristics of the human head.

To enable stimulation of the utricle, slow tilt of the patient at a rate below the threshold for stimulation of the semicircular canals (one to three degrees per second per second) is presently used. Other methods of stimulation specific to the utricle (such as rectilinear acceleration and centrifugal force) will also be used but will be

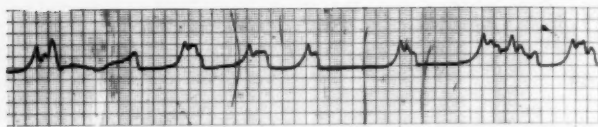


FIGURE 6

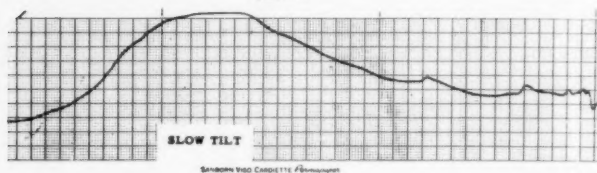


FIGURE 7

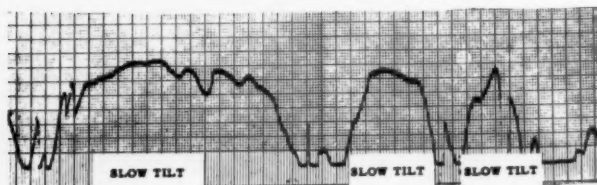
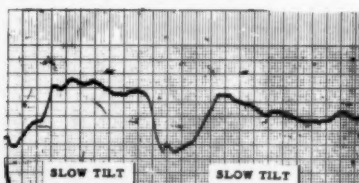


Figure 8

reported at a later date. Suffice it to say here, that no matter just what controlled method of utricular stimulation is used, eye deviation measurements serve as a specific means of measuring the responses both quantitatively and qualitatively (as indicated by the few experiments which have been done in this regard by Versteegh,⁸ Tait and McNally,⁷ and Sullivan, Johnson and Smith.⁶

RESULTS

In Figure 3 there is shown the result of a typical calibration procedure which is routinely carried out to accurately determine the number of degrees of eye movement, either horizontally or vertically as the case may be, which will correspond to any amplitude of deflection on the recording. To enable this, the patient is asked to fix his vision on a small black dot on the wall ten feet away and in line with the relaxed field of vision. He is then instructed to shift his line of sight to another target one foot away from the previous one and located either laterally or vertically with respect to the previous one. This causes a rotation of the eyeball of one degree. The sensitivity of the instrument is simply adjusted at this time to give an appreciable deflection which means that it is now ready to measure the most minute eye movements.

When vestibular stimulation is initiated, the room is either darkened or the patient's head covered over so as to minimize eye movements due to extr vestibular causes.

The high sensitivity of the instrument is exemplified in Figure 4 which shows a very fine nystagmus not perceptible to the examiner by direct visual examination of the eye.

In Figure 5 is seen a normal nystagmus also the result of caloric stimulation. Examination of this recording indicates the typical "saw-tooth" characteristics with the consistently uniform gentle slope being due to the labyrinthine response.

In Figure 6 is seen a pathological complex in a labyrinthine response to caloric stimulation. The patient, a 23-year old man, was referred because of complaints of paroxysmal dizziness accompanied by inability to focus clearly on surrounding objects. Although caloric stimulation showed equal left and right-sided vestibular sensi-

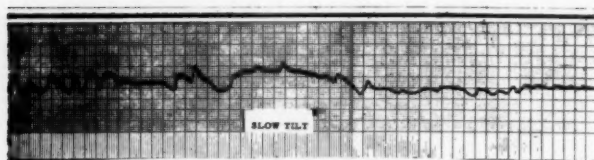


FIGURE 9

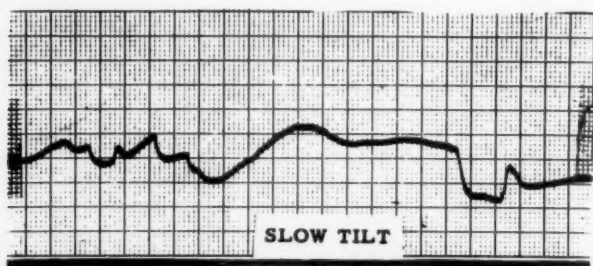


FIGURE 10

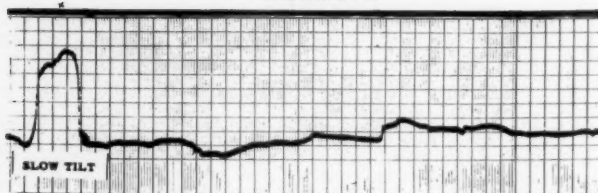


FIGURE 8

tivity with the nystagmus lasting for a normal period of time, the qualitative abnormalities seen in the above recordings were suggestive of an intracranial lesion. This was subsequently verified by other neurological findings (temporal lobe epilepsy).

In regard to recordings of activity of the utricular macula, in Figure 7 we see a typical normal response to slow tilt below the threshold of semicircular canal stimulation. Examination of this recording shows a smooth uniform response in that the eyes deviated upward as the head tilted forward and downward while the same

result occurred in the opposite direction in response to backward and upward tilting of the head.

In Figure 8 we see the recording of utricular stimulation in a patient with Ménière's disease. By contrast with the uniform response of the normal as seen in the preceding figure, we noted two obvious characteristics, namely, 1) an irregular response, and 2) the response became fatigued when the test was repeated, a finding which certifies that the lesion is peripheral.⁴

Figure 9 shows the eye movements of a patient ten days post-operatively following a unilateral labyrinthectomy carried out for the relief of incapacitating Ménière's disease. It can be assumed that degeneration of the VIII cranial nerve on the involved side was in an active stage at this time and hence spontaneous irritation of both the utricular and semicircular canal nerve components would result in spontaneous manifestation of the activity of all parts of the membranous labyrinth. Typical nystagmoid movements were consequently recorded with the patient at rest (sitting position). These are superimposed on the utricular response (of contralateral side) to slow tilt and it is interesting to note that the spontaneous nystagmus increase in amplitude after cessation of the utricular stimulation. It may thus be that the contralateral utricle reinforces semicircular canal stimulation. However, further evidence in this regard should be sought.

Figure 10 shows the response to slow tilt of a 52-year old woman who had the symptoms of Ménière's disease for the past two years. At the time of recording, the disease was subjectively in a quiescent state. No gross irregularities of response due to slow tilt are evident.

Figure 11 shows a very marked deviation of the eyes in response to slow tilt in a 54-year old patient recovering from toxic labyrinthitis due to cholecystitis. This can be classified as an exaggerated response with some irregularity at extreme extent of the eye deviation.

COMMENT

Tribute should be paid to contributions of others in attempts to measure utricular activity:

a) The classical experiments of Tait and McNally⁷ still remain as a most outstanding contribution to our knowledge of the functions of the utricular macula. Compensatory movements of various parts of the body of the frog, including eye movements with and without utricular nerve section, were studied in detail by very accurately controlled experiments.

b) Lorento de No⁵ noted deviation of the eyeballs when a rabbit was rotated at constant angular velocity but with the head in an eccentric position. The resultant between the outward centrifugal force and the downward force of gravity produces eye muscle responses from labyrinthine (utricular) stimulation. This has been applied to humans on the U.S. Navy centrifuge by Graybiel and Niven (1953). Their excellent studies in this regard have added greatly to our knowledge of the utricular macula (subjects rotated below threshold of semicircular canal stimulation).

c) At Utrecht in Holland, Von Egmond, Groen and de Witt (1953) have devised a position chair to stimulate the utricle. By firmly fixing a test subject in a ship's stretcher within a system of gimbal rings, the body is slowly tilted either forward or backward (25°). The resulting deviation of the eyes due to otolith stimulation is recorded by having the subject trace on a blackboard with a piece of chalk the position of a visual after-image.

d) A four-pole parallel swing has been used by Hulk and Henkes³ in clinical practice as a means of stimulating the otoliths. Their studies, however, concerned retinal artery responses.

Many more recordings are required before a significant correlation between the various types of vestibular disease and the responses of the utricle can be fully relied upon. The results to date, however, recommend that this new method of diagnosis merits considerable attention. Further conclusions will be presented at some other time.

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REFERENCES

1. DeWitt, G.: Seasickness. *Acta Oto-Laryngologica*, Suppl. 108, 1953.
2. Graybiel, Ashton: The Importance of the Otolithic Organ in Man Based upon a Specific Test for Utricular Function. *Trans. Amer. Otol. Soc.*, Vol. 44, May 1956.
3. Hulk, J., and Henkes, H. E.: *Practical Otolaryngol.* 12:2:65, 1950.
4. Johnson, W. H., and Smith, J. K. B.: Differentiation of Intracranial and Peripheral Vestibular Lesions. *Laryngoscope* 67:7 (July) 1957.
5. Lorento de No., R.: Die Grundlagen der Labyrinth Physiologie. *Skandinav. Arch. f. Physiol.* 45:251, 1926.
6. Sullivan, J. A., Johnson, W. H., and Smith, J. K. B.: Normal and Abnormal Activity of the Human Otoliths. *ANNALS OF OTOLGY, RHINOLOGY AND LARYNGOLOGY* (Dec.) 1957.
7. Tait, J., and McNally, W. J.: Some Features of the Action of the Utricular Maculae of the Frog. *Phil. Trans. Roy. Soc., London, Series B*, 224:513:241-286 (Dec.) 1934.
8. Versteegh, C.: Ergebnisse partieller Labyrinthexstirpation bei Kaninchen. *Acta Oto-laryn.* 11:393-408, 1927.

XXXIV

STUDIES CONCERNED WITH
TUBOTYMPANITIS

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Numerous excellent studies in the literature deal with the biochemical, microbiologic and cytologic changes found in patients with diseases of the eustachian tube and pneumatic spaces of the mastoid process of the temporal bone. Nevertheless, there is no clear understanding of the etiology or pathogenesis, and much disagreement exists regarding the chemical and pathological alterations which take place in tubotympanitis.

With the aid of newer and more refined chemical, histochemical and pathologic techniques, a co-ordinated clinical and experimental laboratory study has been planned which it is hoped will provide additional definitive findings.

This first report will deal primarily with biochemical, cytologic and microbiologic studies in spontaneous human tubotympanitis.

The most perplexing problem encountered in a review of the available literature is the confusing variety of terms used to describe the various types of effusions. Many investigators have attempted to cope with the problem of terminology and have written of catarrhs of the middle ear, acute and subacute catarrhal otitis media, mild

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secretory otitis media, serous otitis, mucous otitis, otitis media with effusion, hydrotympanum and tubotympanitis.¹⁻³

In most instances, the otological findings and the physical characteristics of the effusion have been used as a basis for classification. Although a number of authors have carried out certain chemical analyses on middle ear fluids, there have been only a few attempts actually to classify them on this basis. Schlender,⁴ for example, distinguished transudates from exudates with the criterion that effusions with greater than 3.95% protein were exudates and those with less than 3.95% were transudates.

Ivstam⁵ divided middle ear effusions into serous and mucous types on the basis of their hexosamine and their fucose content.* He classified a specimen as serous when its hexosamine or fucose value was not greater than that in the serum of the same patient, and as mucous when it was. Any hexosamine or fucose present in an effusion in concentration above that found in serum was considered as a measure of mucus.

Other investigators have also utilized the chemical and physical properties of middle ear effusions in an effort to classify their cases. Robison⁶ separated "subacute catarrhal otitis media and mastoiditis with effusion" into two types: one in which the effusion "appeared to be a symptom" containing 9.95% protein and the second in which the effusion "appeared as a clinical entity" and contained 1.59% protein. Hoople⁷ and Tremble⁸ stated that thin serous effusion had a high protein content, and thick mucous effusion a low one. Suehs⁹ found no correlation between total protein content and the cell count in effusions, but he thought that thin serous fluid with a lower cell count tended to have less protein than thick, tenacious fluid which contained many inflammatory cells.

Siirala and Vuori,¹⁰ in agreement with Schlender, Robison, and Suehs, found that the total protein content of effusions increased with the intensity of the inflammation. Carlson and Lökk¹¹ also

* Hexosamine is composed of an amino group attached to a hexose, either glucose or galactose. It is one of the characteristic products of the hydrolysis of glycoproteins, which would include mucins, heparin, mucoproteins, etc. Small amounts are normally present in all serum protein fractions; but a marked increase in concentration is found in certain pathological conditions. Fucose is a methylated pentose which is also a product of glycoprotein hydrolysis.

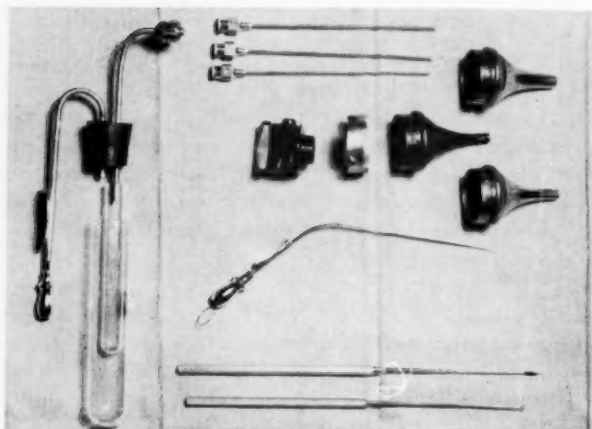


Fig. 1

measured total protein and protein fractions in middle ear effusions and blood serums. They reported that, in 16 out of 17 cases, the total protein concentration in the "transudate" (a term they used for all of their effusions) was greater than that in the corresponding serum.

Many factors have been considered to play a part in the development of effusions. A few investigators have reported finding viruses in association with middle ear disease. Ullman¹² and Wirth¹³ were successful in transmitting to animals the virus found in myringitis bullosa, whereas Senturia and Sulkin¹⁴ were unable to show an increased blood titer of influenza A or B antibodies in this disease. Only recently has an A prime influenza virus been clearly identified in secretions obtained from acute influenzal otitis media.¹⁵

Unsuccessful attempts to isolate a virus from middle ear effusions were made by Forschner,¹⁶ Robison and Nicholas,¹⁷ Siirala,¹⁸ and Harcourt and Brown.² Experiments to identify inclusion bodies in the cells present in middle ear effusions have also been fruitless.¹⁹⁻²⁰

There is general agreement that purulent otitis media is most frequently secondary to bacterial infection,²¹⁻²⁵ but many investigat-

ors were unable to culture bacteria from mucoid and serous effusions, or considered the growth obtained as contaminants.^{7-9,17,26-29} Others reported positive cultures in a small percentage of their cases.^{16,30} Siirala¹⁸ felt that poor bacteriological technique would not explain the small number of positive cultures and showed that middle ear fluid contained bacteriostatic and bactericidal components.

An occasional reference is made to the discovery of bacteria on direct smear of middle ear effusions.^{31,32} King²⁶ found no bacteria on smears in his 20 cases designated as "thin fluid" but reported five smears positive for organisms in a similar number of "thick fluids." In those with bilateral ear involvement, four of the "thick" and none of the "thin" showed bacteria in the 15 cases studied. He described a "large Gram-positive diplococcus" present in four cases and the same organism in combination with a mixed flora of other bacteria in three. Gram-negative rods and diphtheroids were apparent in two instances. Blegvad³² recorded the findings of bacteria both on smear and in culture in 26 out of 53 cases. In seven, bacteria were observed on smear but not recovered in cultures, while in 11 cases organisms were cultured from the secretions but were not seen in direct smears.

Because of the frequent absence of micro-organisms and cellular constituents, as well as the insidious development of many effusions, a diagnosis of allergic effusion is often made. Furthermore, some confusion has resulted from combining under one title of chronic allergic otitis media, those ears with middle ear effusion and an intact drum and those with a perforated tympanic membrane and chronic otorrhea.

Dohlman^{33,34} and Koch³⁵ have written of allergic changes in the chronic draining middle ear. Dohlman emphasized the importance of an allergic evaluation in patients with nonresponsive chronic otitis media and demonstrated eosinophiles in secretions. Koch found a significant number of eosinophiles and changes compatible with the allergic state in 19.8% of his 262 cases.

Lewis³⁶ and Proetz³⁷ suggested that localized allergic edema in the tympanum could simulate acute purulent otitis media. Ojala and Palva³⁸ observed 124 cases of acute and chronic otitis media and mentioned only one instance of apparent allergic origin. Derlacki³⁹ was of the opinion that "the majority of the cases of chronic secretory

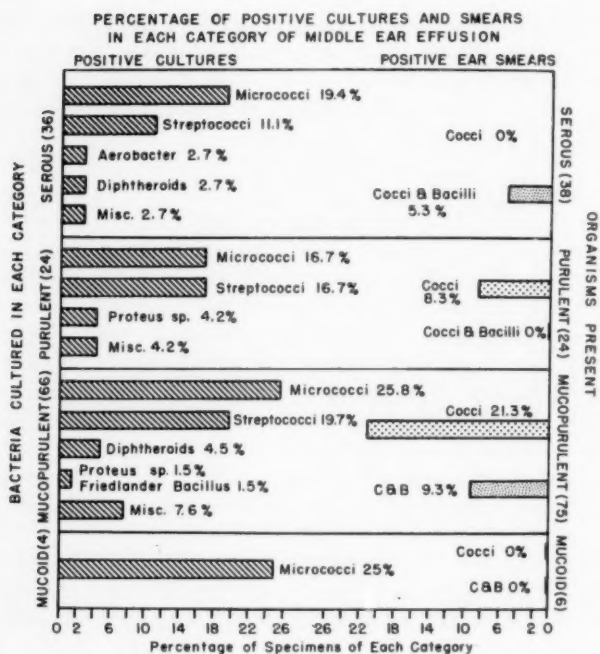


Fig. 2

otitis with either a clear serum or a much thicker mucus were due to a definite allergy to house-dust or to a food." He described the characteristic picture of allergic chronic otitis as follows:

1. Viscous or gelatinous secretion with a considerable number of eosinophiles.
2. Thickened mucoperiosteum (like allergic mucous membrane).
3. Protracted healing.
4. Frequent occurrence in allergic individuals.
5. Good response to anti-allergic treatment.

Jordan³⁰ described 123 patients with middle ear effusion among which 91 (74%) were diagnosed as due to allergy on the basis of eosinophiles in nasal smears, skin tests and response to allergic therapy. On direct smear of the effusion only an occasional eosinophile was found. King²⁶ studied the effusions obtained from 72 ears of 56 children. He reported that 7 of the 39 ears with mucous secretion show 5 to 10% eosinophiles in the secretion while none of the children with serous effusion had any eosinophiles. Ivstam⁵ recorded smears from 33 cases with secretory catarrh and 13 of these showed eosinophiles in very small numbers.

Hotchkiss³¹ stated that the eosinophile count in middle ear effusions was not dependable. Suehs⁴⁰ in 1952, saw only one eosinophile in some 50 slides examined among his 372 cases. Robison and Nicholas¹⁷ found no eosinophiles in a hundred cases of acute and chronic otitis media and secretory catarrh. In some 50 cases of serous otitis studied by the Bryans⁴¹ all smears were negative for eosinophiles.

Most investigators are willing to recognize the relationship of sinus and nasopharyngeal infection to the development of tubal and tympanic disease. Robison has quoted in detail the findings of Rouviere⁴² regarding the lymphatics of the eustachian tube and the nasopharynx and has suggested that the pretubal plexus, retropharyngeal lymph nodes and the upper cervical glands play an important role in the development of middle ear effusions.⁶

Rüedi^{43,44} described in great detail the histologic structure of the mucoperiosteal lining of the pneumatic spaces of the mastoid process, while Beck⁴⁵ and Singer⁴⁶ suggested that the membrane lining the tympanum may under certain conditions acquire the ability to secrete mucus. Friedmann⁴⁷ and Ojala⁴⁸ demonstrated the presence of mucus secreting glands in the middle ear "mucosa" of humans, while the former⁴⁹ successfully produced secretory glands in membrane lining the guinea pig bulla. Friedmann's studies appear to confirm the theory that inflammation may cause a reversion of the mucoperiosteum to a secretory mucous membrane.

In recent years a good deal of attention has been given to the nature of the cells found in middle ear effusions. There are numerous reports of finding neutrophils, a small number of lymphocytes and mononuclear cells in addition to occasional eosinophiles in middle ear

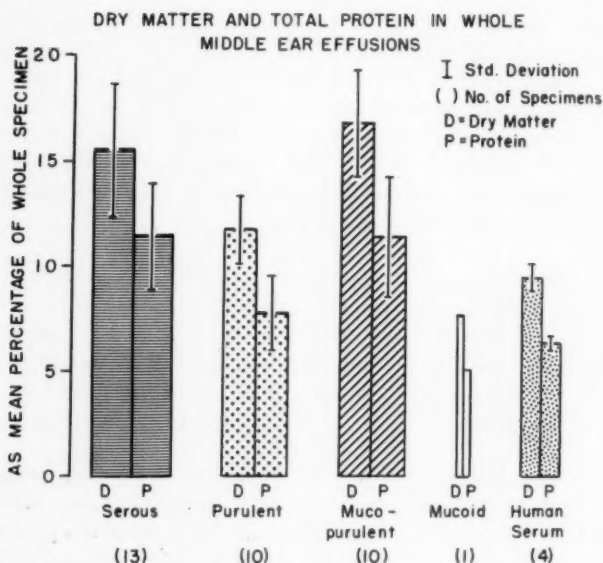


Fig. 3

effusions. Forschner¹⁶ noted free nuclei in addition to the other cells present. Löwy⁶⁰ observed that there was great variability in the number of cells and was able to count as many as 100 neutrophils per cubic millimeter. More recently, Robison⁶ divided the cases into an exudative type, rich in neutrophils, and a transudative type having only a few neutrophils. He stated that when the chronic catarrhal stage of the disease was reached, large mononuclear leukocytes were found in the secretion. Jordan²⁰ described scattered neutrophils and an occasional eosinophile in serous type cases. King²⁶ observed little difference in the distribution of neutrophils in the specimens examined. Suehs⁹ indicated that the thin serous cases contained only a few neutrophils whereas the tenacious fluid showed as many as 300 per high power field. Davison²⁷ stated that there were present in the mucoid type, a varying number of neutrophils and a few macrophages. Ivstam⁵ reported that the mucoid secretion

in children usually contained cells. The Bryans⁴¹ found a number of serous specimens with lymphocytes and only a few with neutrophils and phagocytes. Bryan⁵¹ and Ojala and Palva³⁸ described large phagocytes whose function in aural secretions is not known.

EQUIPMENT, METHODS AND TECHNIQUES

The special equipment used in these studies consisted of aspirating sets (Fig. 1) in covered stainless steel basins which were sterilized by autoclaving. These sets were composed of the following:

1. Myringotomy knives.
2. Ear speculae.
3. Metal, ringed ear curette.
4. Aural magnifier (Bruening-Work).
5. Adapter for otoscope speculae.
6. Aspirators, consisting of a large glass tube (18x90 mm) into which was placed a 3 ml glass tube to catch the secretion, a Luer-lok tip to accommodate needles and a suction inlet and outlet.
7. Needles, three-inch, short bevel, 16, 17, 18 gauges.

At the time of surgery, the ear canals were cleaned of any accumulated cerumen but no attempt was made to sterilize the meatus or the tympanic membrane.

Under general anesthesia and sterile operating room conditions, the tympanic membrane was incised, an appropriate gauged needle inserted into the middle ear and the secretion aspirated into the 3 ml tube contained within the larger glass tube.

When thin, serous secretions were collected, the procedure was accomplished in a matter of seconds and a minimum of fresh blood was mixed with the fluid. Where some difficulty was encountered in removing the more viscous secretions, and in purulent otitis media a small bloody admixture frequently occurred.

The aspirator and its contents were transferred to the laboratory within a period of 15 to 30 minutes after the conclusion of the oper-

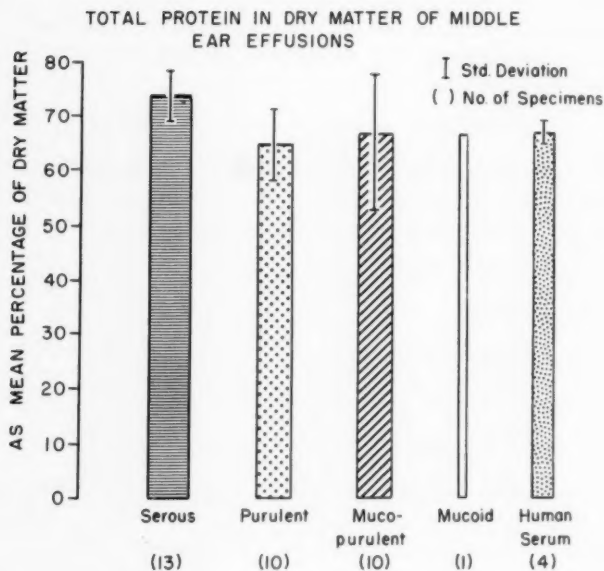


Fig. 4

ative procedure. After gently stirring the specimen with a sterile 3 mm platinum inoculating loop, in order to obtain a uniform mixture, a small portion was removed for cytological and bacteriological studies. The remainder was stored in a frozen state until the chemical determinations were performed.

The material for microscopic study was spread on two slides over an area of approximately 0.5 x 1.5 cm. These were stained according to the Papanicolaou and Wright's techniques.

One loopful of the middle ear fluid received for culture was routinely inoculated into fluid thioglycollate broth, for the initial cultivation of both aerobic and anaerobic micro-organisms. The Spray dish, with pyrogalllic acid and sodium hydroxide, was used for the cultivation of anaerobes. When growth was apparent, the organ-

isms were subcultured onto appropriate solid media, in most instances blood agar plates. (Sterile whole citrated human and defibrinated rabbit blood was used in the preparation of the blood agar plates.)

The Streptococci cultured were grouped according to their reaction on blood and no further attempt was made to classify them.

A small number of the specimens were positive for growth in the thioglycollate broth, but repeated attempts to subculture the bacteria by aerobic and anaerobic methods failed. These bacteria, on smear, were Gram-positive cocci, either in pairs or in short chains. Cultures were reported negative only after absence of growth for seven days.

The following values of cytologic elements per high power field were used in order to provide some gross quantitative method of interpreting the findings:

NEUTROPHILES, LYMPHOCYTES, CELLULAR REMNANTS AND BACTERIA

\pm	1 to 10
+	11 to 20
2 +	21 to 50
3 +	51 to 75
4 +	> 75

EOSINOPHILES AND PHAGOCYTES

\pm	1 to 5
+	6 to 10
2 +	11 to 15
3 +	16 to 20
4 +	> 20

MUCOUS STRANDS

Occasional
Moderate
Numerous

For the biochemical analyses, specimens were removed from the deep freeze and thawed. No cellular material was removed by centrifugation, but rather the whole specimen was used for the following

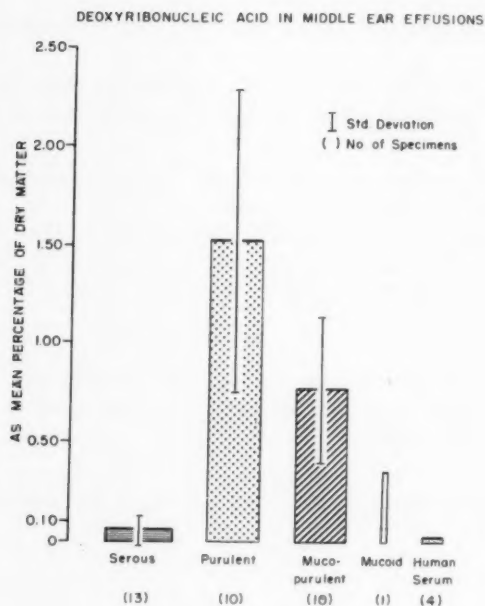


Fig. 5

determinations: total dry matter, total protein, deoxyribonucleic acid (DNA) and protein-bound carbohydrate. DNA is the component of nucleo-proteins which is found primarily in cell nuclei. Very little of it is to be found in serum, since the white cells should be its only source in whole blood; erythrocytes do not normally contain any DNA. In this study the protein-bound carbohydrate is that which precipitates together with the protein in ice-cold perchloric acid, and can be hydrolyzed and extracted from the precipitate with hot perchloric acid. It was measured as glucose.

The dry matter was measured as a percentage of the whole specimen on a weight per volume basis by Lowry's micro-method.⁵² Protein, DNA and protein-bound carbohydrate were each reported as a percentage of the dry matter and were also calculated as a

percentage of the whole specimen where these figures were of interest. The colorimetric technique used for protein was that of Lowry et al.,⁵³ DNA was determined by the fluometric method of Kissane and Robins,⁵⁴ and protein-bound carbohydrate was measured by a modification of Goa's method.⁵⁵ Complete details concerning the application of these procedures are reported elsewhere.⁵⁶

RESULTS

There was a difference between the number of patients examined and the number of specimens studied since more than one specimen was obtained from a few patients and not all specimens were examined bacteriologically and cytologically. Chemical analyses were performed on a small number of selected specimens. The basic figures are assembled in Table I.

TABLE I
SUMMARY OF PATIENTS AND SPECIMENS EXAMINED

Patients with Middle Ear Effusions	105
Specimens of Middle Ear Effusions	150
Specimens Examined Cytologically	143
Specimens Examined Culturally	130
Specimens Examined Chemically	34

On the basis of history, otologic findings and research data which will be presented in the latter part of this paper, a working nomenclature for middle ear effusions has been evolved (Table II).

The term serous otitis was limited to those forms of catarrhal otitis or secretory otitis in which the effusion consisted of a very pale straw to a salmon-colored, low viscosity secretion which had a tendency to gel when exposed to air.

In the purulent category were placed the classical cases of suppurative otitis media. The secretion was grey-white in color, slightly increased in viscosity, homogeneous and turbid. On standing, there was a tendency for the cellular constituents to settle out so that a bi-phasic appearance developed.

TABLE II
NOMENCLATURE OF MIDDLE EAR EFFUSIONS

DIAGNOSIS	SYNONYMS
1. Serous	Secretory otitis media Otitis media with effusion Catarrhal otitis Hydrotympanum
2. Purulent	Acute otitis media Suppurative otitis media
3. Mucopurulent	Chronic otitis media Secretory otitis media Glue ear Exudative catarrh Catarrhal otitis Mucoid ear
4. Mucoïd	Glue ear Allergic otitis media Secretory otitis media

The mucopurulent specimens consisted of a dirty-grey, translucent, highly viscid, sticky, homogeneous material; occasionally interspersed was a brownish, semi-opaque material.

The mucoïd effusions were grossly similar to the mucopurulent, except that they had an extremely rubbery consistency. It was difficult to distinguish the mucoïd from the mucopurulent without laboratory assistance.

TABLE III
CELLULAR AND MUCOID ELEMENTS IN MIDDLE EAR EFFUSIONS

	MUCOUS STRANDS	NEUTRO- PHILES	LYMPH- OCYTES	EOSINO- PHILES	PHAGO- CYTES	CELLULAR REMNANTS
Serous	Occasional	±	±	0	± to +	±
Purulent	Occ. to Num.	+ to 4+	± to 2+	±	± to +	± to 2+
Mucopurulent	Occ. to Num.	±	±	0	± to 2+	2+ to 4+
Mucoïd	Numerous	0	0	0	±	±

A differentiation of all four categories of effusions was based on the cytologic findings, as shown in Table III. Bacteriologic findings on both smears and cultures are summarized in Table IV.

TABLE IV
PERCENTAGE OF POSITIVE CULTURES AND SMEARS
IN VARIOUS CATEGORIES

PERCENTAGE OF SPECIMENS				
CATEGORY	NUMBER OF SPECIMENS	POSITIVE CULTURES	BACTERIA PRESENT IN SMEAR	BACTERIA PRESENT IN SMEAR BUT CULTURE NEGATIVE
Serous	36	33.3	5.3	2.8
Purulent	24	29.1	8.3	4.2
Mucopurulent	66	51.5	30.6	10.6
Mucoid	4	25.0	0.0	0.0

In the serous specimens, there were only a few neutrophiles and lymphocytes present. Large phagocytes were seen in some instances and a very occasional eosinophile was noted. Rare nuclear remnants were observed. One-third of the specimens gave positive cultures showing primarily streptococci and micrococci. A few of the smears (5.3%) revealed a very small number of cocci and bacilli.

The purulent specimens, on microscopic examination, showed a moderate number of mucous strands, myriads of intact neutrophiles, occasional large and small phagocytes, a few lymphocytes, a rare eosinophile and a moderate number of nuclear remnants. On culture slightly less than one-third of the specimens were positive for micrococci and streptococci (alpha and gamma) while 8.3% of the smears showed cocci.

Cytologic examination of the mucopurulent effusions usually revealed myriads of mucous strands and large numbers of nuclear remnants in various stages of degeneration. These included some neutrophilic nuclear remnants, but the largest percentage were of mononuclear type. A few intact neutrophiles were found. Two slides of 75 examined showed a rare eosinophile. A positive bacterial

culture was obtained in over one-half of these specimens. It is noteworthy that 30.6% of the specimens revealed micro-organisms on smear and that 10.6% showed bacteria without positive cultures.

The mucoid specimens presented myriads of mucous strands, rare nuclear remnants, neutrophils, lymphocytes, and phagocytes. No eosinophils were seen. There were insufficient specimens to interpret either the cultures or smears of the mucoid type.

TABLE V
NUMBER AND PERCENTAGE OF VARIOUS BACTERIA
CULTURED FROM MIDDLE EAR EFFUSIONS

ORGANISMS	NUMBER	PER CENT
<i>Micrococcus albus</i>	20	30.2
<i>Micrococcus albus</i> , hemolytic	3	4.6
<i>Micrococcus aureus</i>	4	6.1
<i>Micrococcus</i> , undifferentiated	3	4.6
<i>Streptococcus</i> , α -type	7	10.6
<i>Streptococcus</i> , β -type	2	3.0
<i>Streptococcus</i> , γ -type	3	7.6
<i>Streptococcus</i> or <i>Pneumococcus</i>	7	10.6
<i>Diphtheroids</i>	4	6.1
<i>Bacillus</i> sp.	7	10.6
<i>Klebsiella pneumoniae</i>	1	1.5
<i>Proteus</i> sp.	2	3.0
<i>Aerobacter aerogenes</i>	1	1.5
Total	66	100.0

Cultures were obtained on 130 of the collected specimens and bacterial growth was observed in 54 (41.5%). More than one organism was found in some specimens so that 66 positive growths were examined. Eighty-eight per cent of the positive cultures were aerobes, 9% facultative anaerobes and only 3% were anaerobes. The micro-organisms cultured are shown in Table V and in Figure 2.

Analyses were made on 34 specimens of middle ear effusions in order to discover possible biochemical differences between the various categories. These included 13 of the serous category, ten purulent,

ten mucopurulent and one mucoid. The same analyses were made on four human blood serum specimens.

The results of the biochemical determinations are presented in Tables VI and VII and are shown graphically in Figures 3 to 6. The

TABLE VI

DRY MATTER AND PROTEIN IN WHOLE MIDDLE EAR EFFUSIONS
(As mean percentages of whole specimens \pm standard deviations)

CATEGORY	NUMBER OF SPECIMENS	DRY MATTER	PROTEIN
Serous	13	15.5 \pm 3.2	11.4 \pm 2.6
Purulent	10	11.8 \pm 1.6	7.7 \pm 1.7
Mucopurulent	10	16.8 \pm 2.4	11.4 \pm 2.8
Mucoid	1	7.7	5.1
Serum	4	9.4 \pm 0.6	6.3 \pm 0.4

TABLE VII

VARIOUS COMPONENTS IN THE DRY MATTER OF EFFUSIONS
(As mean percentages \pm standard deviations)

CATEGORY	NUMBER OF SPECIMENS	PROTEINS	DEOXYRIBO-NUCLEIC ACID	PROTEIN-BOUND CARBOHYDRATES (HEXOSE)
Serous	13	73.5 \pm 4.6	0.05 \pm 0.07	0.53 \pm 0.26
Purulent	10	64.4 \pm 6.3	1.52 \pm 0.76	0.79 \pm 0.35
Mucopurulent	10	66.1 \pm 10.9	0.77 \pm 0.36	3.07 \pm 1.35
Mucoid	1	65.7	0.36	0.43
Serum	4	66.5 \pm 2.0	0.01 \pm 0.00	0.35 \pm 0.06

determination of total dry matter in various categories of whole effusions revealed that the mucopurulent and serous specimens contained higher percentages of dry matter than purulent (Fig. 3). The difference between the percentage of dry matter in the mucopurulent (16.8%) and serous (15.5%) was not significant, but both of these means were significantly greater than that of the purulent category (11.8%). The means were compared by the "t" test and were

considered to be significantly different if "P" was less than 0.01.⁵⁷ In many instances "P" was actually less than 0.001. Dry matter in the purulent was in turn slightly higher than the dry matter in serum (9.4%). The one mucoid specimen contained less dry matter (7.7%) than serum. Although all of the results on this single specimen were included in the tables and figures, no interpretation nor extrapolation of these findings to apply to the entire mucoid category was attempted.

The total protein content was reported both as a percentage of the whole specimen and of the dry matter. When it was considered as a percentage of the whole (Fig. 3), the mean values for mucopurulent and serous effusions were the same (11.4%). The protein in whole purulent (7.7%) was significantly less, but still greater than the mean for four serum specimens (6.3%). The mucoid effusion contained less protein (5.1%) than serum.

When protein was considered as a percentage of the total dry matter (Fig. 4), then the group means were much closer among mucopurulent (66.1%) and purulent specimens (64.4%) and serum (66.5%). The only mean which was significantly higher on this basis was that for serous effusions (73.5%) as compared to those for purulent effusions and serum. The one mucoid value (65.7%) was very close to the mean for mucopurulent.

Of those biochemical constituents which were measured, the deoxyribonucleic acid (DNA) showed the greatest differences in mean concentrations in the various categories of effusions (Fig. 5). On a dry weight basis, the mean value for purulent specimens (1.52%) was twice as large as the mean for mucopurulent effusions (0.77%) and about 30 times more than that for the serous category (0.05%). The mucoid specimen (0.36%) showed about half as much DNA as the mucopurulent group, but considerably more than the serous group and serum (0.01%), which contained almost no measurable amount. The mean concentrations of DNA in both the purulent and the mucopurulent categories were significantly greater than the means for the serous specimens and for serum. The serous group was not significantly different from serum. In the findings regarding both DNA and protein-bound carbohydrate, there was considerable variation within each category (as shown by the rather large standard deviations reported in the tables and graphs), but

this did not prevent significant differences between some of the means from showing up.

The mean protein-bound carbohydrate (as a percentage of dry matter) in the mucopurulent specimens (3.07%) was about six times as much as in serous effusions (0.53%), almost four times as much as in purulent (0.79%), and about nine times as much as in serum itself (0.35%) (Fig. 6). The protein-bound carbohydrate in the one mucoid (0.43%) was less than the mean for serous specimens. The mean for the mucopurulent category was significantly higher than the means for all other groups. It was also found that the mean for the purulent category was significantly higher than that for serum, but not for serous specimens. The concentration of protein-bound carbohydrate in serous effusions was not statistically different from that in serum.

After the microscopic studies were performed and the classification of specimens was established, all the specimens were divided into categories according to the cytologic findings. The distribution of ages and effusions are shown in Figures 7 and 8. The largest number of specimens examined were the mucopurulent (50%) while the serous constitute only 26%. A much smaller number of mucoid (6.7%) and purulent specimens (17.3%) were included (Fig. 7).

It was noted that the greatest number of mucopurulent and purulent specimens fell into the two to eight year age groups while the serous specimens were obtained over a wide age range with some predominance in the groups below eight and over thirty years (Fig. 8). The mucoid group appeared to have a fairly even distribution.

COMMENT

In pursuing the over-all plan of this investigation, it was recognized that, on the whole, there would be better control and follow-up on patients seen in private practice. Therefore most of the specimens in this study were obtained from private patients. A small number were submitted from the outpatient department of Washington University Clinics.

The age distribution of patients and the category breakdown of specimens were influenced by the fact that a large number of adults

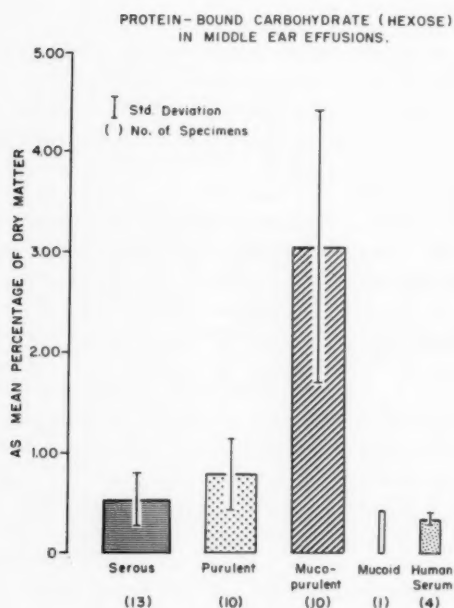


Fig. 6

refused general anesthesia, had myringotomies in the office and the specimens were not submitted for study. In addition, since the serous fluid in many of the adults and some of the children could be evacuated via the eustachian tube, a smaller percentage of serous cases than were actually seen were included in this study.

All the children seen in the office with persistent effusions were required to have myringotomies under general anesthesia. Only in the latter part of the study was it decided to obtain a small number of specimens of acute purulent otitis media in order to compare the findings with the other categories.

As a consequence of these conditions, it cannot be said that this series of specimens constitute a random selection of patients or

represents a true cross section of acute and chronic middle ear effusions seen in private practice.

When aspirating the fluid, the gross physical characteristics provided clues as to the category of effusion. This was especially true if the effusion was typically serous. On the other hand, we were not able to predict accurately the microscopic findings of the purulent, mucopurulent or mucoid type. Since the best therapeutic results will be obtained if it is possible to demonstrate the presence or absence of inflammatory process, it is of prime importance that specimens be collected and examined microscopically and bacteriologically.

There has been a great difference of opinion regarding the significance of positive bacterial cultures obtained from the effusions. In considering our own data we are well aware that the failure to sterilize the ear canal may have increased the percentage of positive cultures. This was a calculated risk taken because one of our primary objectives was to obtain satisfactory samples of effusions for chemical analysis and any antiseptic placed into the canal might have influenced the highly sensitive microchemical determinations.

We are of the opinion, however, that the confirmatory evidence presented by the high percentage of bacteria found on direct smears made from the aspirated secretion is of particular importance. These bacteria must have been present prior to myringotomy since there was insufficient time for any contaminant to have multiplied sufficiently to produce the bacteria found on the slide.

When one correlates the high percentage of bacteria on smears, the positive cultures and large number of inflammatory cells, all of which are present in the mucopurulent category, it appears strikingly evident that the mucopurulent secretion is of inflammatory origin.

The findings regarding dry matter content demonstrate that the viscosity and general physical properties of the effusions are not dependent upon the total content of dry matter. Mucopurulent and serous effusions, which differ greatly in physical properties, have almost the same total dry matter, whereas the purulent, which are intermediate between the other two with regard to viscosity, have a surprisingly lower content of dry matter.

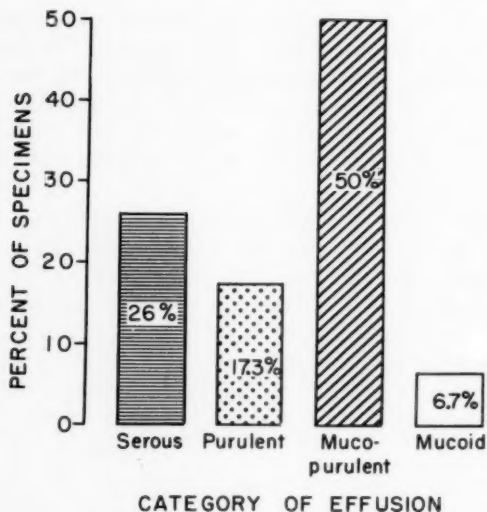
PERCENTAGE OF SPECIMENS IN EACH
CATEGORY OF MIDDLE EAR EFFUSION.

Fig. 7

The average protein content of mucopurulent effusions closely resembles that of serous effusions on the whole specimen basis. It appears from these findings that no clear-cut distinction can be made between the various types of effusions on the basis of their total protein content alone. It may also be stated that the greater viscosity of the mucopurulent specimens as compared with serous effusions is not due to the presence of less water or more total protein.

Reports in the literature regarding the total protein content of the different types of effusions have been conflicting. Our data do not support the contention that the thin serous fluid has a higher protein content than the thick mucous exudate except on a dry weight basis, if by "thick, mucous exudate" one refers to the mucopurulent specimens or so-called "glue ears." Our findings for the

mean protein concentrations in mucopurulent and serous specimens agree closely with the mean (11.4%, w/v.) reported by Carlson and Lökk¹¹ which apparently included both of these groups.

All categories of effusions have a significantly higher average dry matter content than normal serum. If these effusions originate as transudates, then water resorption occurred before the specimens were collected. If this is true, then a distinction cannot be made between transudates and exudates merely on the basis of the percentage of dry matter or total protein in the aspirated whole specimen.

Deoxyribonucleic acids are found in high concentrations in cell nuclei, so the differences in the various categories undoubtedly reflect the differences in the amounts of nuclear material they contain. The purulent specimens, which show relatively large numbers of polymorphonuclear leukocytes, usually with some nuclear remnants in addition, are found to have the highest DNA content. The mucopurulent specimens contain only a few polymorphonuclears, but many nuclear remnants, which could account for the fact that their DNA content is high, but not so high as that of purulent specimens. Serous effusions contain no nuclear remnants and very few polymorphonuclears, hence they contain very low concentrations of DNA. The serous specimens are, in this respect, very similar to serum, which is found to contain almost no measurable DNA. The DNA values for serous effusions are in no cases even close to the values for mucopurulent or purulent samples. There is apparently a very good correlation between the number of cells plus nuclear remnants found cytologically and the amount of DNA found chemically. The correlation could not be determined by statistical methods and expressed numerically because the cytological and chemical data were not of the same nature.

The findings with respect to protein-bound carbohydrate are taken to indicate that the mucopurulent specimens are particularly rich in glycoproteins, in amounts far in excess of those found in any other specimens. The concentration of protein-bound carbohydrate seems to correlate very well with the cytological findings regarding mucous strands. Specimens which are high in mucous strands are also high in protein-bound carbohydrate, and those with few or no mucous strands are low.

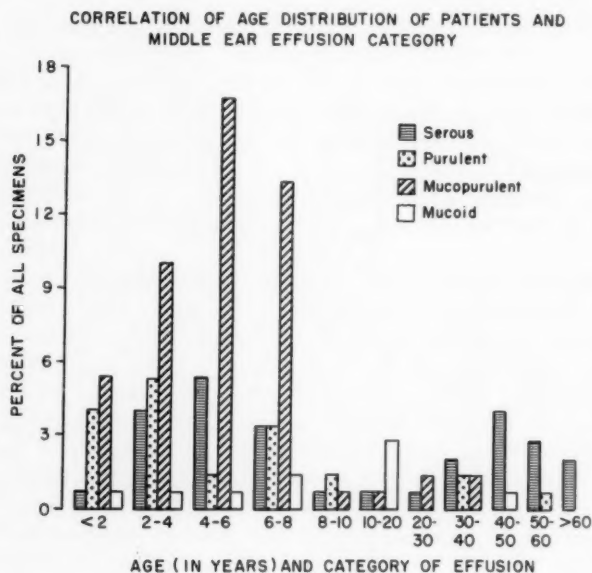


Fig. 8

It is also noted that those same specimens which are rich in protein-bound carbohydrate usually contain an abundance of nuclear remnants, but not a large number of whole polymorphonuclear leukocytes. In other words, evidence for the presence of mucus is the strongest in the effusions which contain products of cellular breakdown. This observation can be interpreted to suggest that the increased amount of glycoprotein in mucopurulent specimens results from some long standing irritation and tissue breakdown of the middle ear lining membrane. However, it could likewise have come from mucus which was secreted by goblet cells or mucous glands within a metaplastic mucous membrane in the middle ear cavity. It is unlikely that the protein-bound carbohydrate could have originated from a transudative fluid, since it is not present in blood serum in sufficient concentration to account for the amounts found in mucopurulent effusions.

Since the actual source of the increased amounts of protein-bound carbohydrate in mucopurulent specimens is still undetermined, further studies are required before an accurate interpretation of its clinical significance can be made.

The positive evidence accumulated regarding the inflammatory nature of the mucopurulent category is the strongest argument against the widely prevalent theory that this effusion is of allergic origin. In addition, there is a lack of clear scientific evidence to support the claim that the fluid is an allergic effusion. Our own observations agree with those of investigators who were unable to find eosinophiles in the fluid obtained from the middle ear.

We readily agree that some patients with middle ear effusions show an allergic diathesis including pale, hypertrophic nasal and sinus mucous membranes, profuse mucoid nasal secretion, and lymphoid hyperplasia of the nasopharynx. These individuals are unquestionably more susceptible to superimposed or secondary infection of the sinuses and middle ears. However, the evidence presented in these studies strongly suggests that therapy should be directed first to the control of the inflammatory process in the nasal fossae, sinuses, nasopharynx and middle ear. If rapid and complete cure does not occur, every effort should be made to discover and remove antigenic factors which are contributing to an allergic state. We believe it is imperative to emphasize the primary control of the inflammatory components and to establish perspective regarding the contributory part played by allergy in the serous, purulent or mucopurulent categories of middle ear effusions.

It would appear, from the review of the literature and our data accumulated up to this time, that the largest percentage of middle ear effusions may be encompassed by the following speculative concept of pathogenesis: during an acute rhinopharyngitis there is obstruction of the eustachian tube and viral or bacterial contamination of the tube and middle ear. An inflammatory (vascular) reaction occurs within the mucoperiosteum and there results an extravasation of certain noncellular constituents of the serum into the lumen of the middle ear. If adequate immunologic defenses are present (as in most adults), the micro-organisms are inactivated or destroyed. After the upper respiratory disease is controlled, some of the aqueous components of the effusion are resorbed by the lymphatics

or blood vessels, leaving a high protein, low carbohydrate (mucus), noncellular (low DNA) effusion which must be evacuated by myringotomy or via the eustachian tube.

In the event that good antibody titers are not present, and antibiotics are not given, the micro-organisms multiply and a cellular (phagocytic) response is evoked, thus producing a purulent otitis media. The effusion would contain many neutrophils (high DNA), moderate protein, low carbohydrate (mucus), and a positive culture and smear. Spontaneous rupture of the tympanic membrane occurs, or myringotomy is performed in this instance to provide drainage of the purulent fluid.

If inadequate antibiotic therapy is given, or the patient presents a delayed immunologic response, subsidence of otologic symptoms may occur without drainage of the exudate from the tympanum. The smoldering subacute inflammation will cause metaplasia of the mucoperiosteal lining of the middle ear, and there will appear goblet cells and mucous glands which secrete glycoproteins. At this stage one would find that the middle ear effusion contains myriads of nuclear remnants, some intact neutrophils, large amounts of carbohydrate (mucus), and a positive culture and smear (the mucopurulent category). A myringotomy and antibiotic therapy may control the inflammatory component of the disease but there will be prompt recurrence of an effusion because of the secretory activity of the metaplastic mucous membrane. Only persistent treatment of the nasopharynx, evacuation of the fluid and prevention of respiratory infections will permit a return of the mucous membrane to its previous non-secretory state.

The foregoing hypothesis presents many known facts as well as much speculation. We are aware of the many unresolved problems. These studies are presented as the first report of our investigations of tubotympanitis. Human and animal biopsies taken from the middle ear are under study. Problems related to the experimental production of tubotympanitis in animals are being solved. Preliminary investigations of the *in vitro* activities of various enzymes upon the complex glycoproteins are now in progress. We hope that we shall have the opportunity of presenting these findings in the near future.

REFERENCES

1. Politzer, A.: A Textbook of the Diseases of the Ear. Lea Bros. and Co., Philadelphia and New York, 1903.
2. Harcourt, F. L., and Brown, A. K.: Hydrotympanum (Secretory Otitis Media). *Arch. Otolaryng.* 57:12-21, 1953.
3. Van Dishoeck, H. A. E.: Hydrotympanum in the Different Clinical Aspects of the Tubotympanitis Catarrh. *Acta Oto-laryng.* 36:429-436, 1949.
4. Schlandler, E.: Zur Pathologie des sekretorischen Katarrhs. *Monatsschr. Ohrenh.* 66:278-285, 1932.
5. Ivstam, B.: On Secretory Catarrh of the Middle Ear with Special Reference to its Pathogenesis and Prognosis. *Acta Oto-laryng.* 44:274-285, 1954.
6. Robison, M. J.: Subacute Catarrhal Otitis Media and Mastoiditis with Effusion. *South. M. J.* 35:815-825, 1942.
7. Hoople, G. D.: Otitis Media with Effusions—A Challenge to Otolaryngology. *Laryngoscope* 60:315-329, 1950.
8. Tremble, E. G.: Secretory Effusion of the Tympanum. *Laryngoscope* 61:791-802, 1951.
9. Suehs, O. W.: Chronic Secretory Otitis Media: Etiology, Diagnosis and Treatment. *J. Med. Assn. Ga.* 45:499-506, 1956.
10. Siirala, U., and Vuori, M.: Protein Pattern and Bacteriostatic Effect on the Exudate in Acute Otitis Media. *Acta Oto-laryng.* 3:197-204, 1954.
11. Carlson, L. A., and Lökk, T.: Protein Studies of Transudates of the Middle Ear. *Scand. J. Clin. Lab. Invest.* 7:43-48, 1955.
12. Ullman, E.: Zur Kenntnis der Myringitis bullosa. *Monatsschr. Ohrenh.* 60:84-87, 1926.
13. Wirth, E.: Studien zur klinischen Bakteriologie der akuten Mittelohrentzündung. *Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres.* 27:21-59, 1928.
14. Senturia, B. H., and Sulkin, S. E.: The Etiology of Myringitis Bullosa Hemorrhagica. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 51:476-482, 1942.
15. Yoshie, E.: On the Isolation of Influenza Virus from Mid-Ear Discharge of Influenza Otitis Media. *Jap. J. Med. Sci. and Bio.* 8:373-377, 1955.
16. Forschner, L.: Über den sogenannten sekretorischen Mittelohrkatarth. *Ztschr. f. Hals-, Nasen- u. Ohrenh.* 12:477-487, 1925.
17. Robison, J. M., and Nicholas, H. O.: Catarrhal Otitis Media with Effusion. *South. M. J.* 44:777-789, 1951.
18. Siirala, U.: The Problem of Sterile Otitis Media. *Pract. Oto-rhin-laryng.* 19:159-169, 1957.
19. Carr, C. D., and Senturia, B. H.: Unpublished data.
20. Bryan, W. T. K., and Bryan, M. P.: Personal Communication.

21. Laikainen, E. A.: Clinico-Bacteriological Studies on Acute Otitis Media. *Acta Oto-laryng. Suppl.* 107:1-82, 1953.
22. Henry, L. D., and Kuhn, H. A.: Bacteriological Studies of Acute Infections of the Middle Ear. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 49:519-525, 1940.
23. Friedmann, I.: Bacteriologic Studies. *J. Laryng. and Otol.* 66:175-180, 1952.
24. House, H. P.: Otitis Media. *Arch. Otolaryng.* 43:371-378, 1946.
25. Eagle, W. W.: Secretory Otitis Media. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 55:55-67, 1946.
26. King, J. T.: The Condition of Fluid in the Middle Ear: Factors Influencing the Prognosis in 56 Children. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 62:498-506, 1953.
27. Davison, F. W.: Secretory Otitis Media. *Bull. Geisinger Mem. Hosp. and Foss Clin.* 6:58-66, 1954.
28. Hoople, G. D., and Blaisdell, I. H.: The Problem of Acute Catarrhal Otitis Media. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 52:359-363, 1943.
29. Hoople, G. D., and Blaisdell, I. H.: Some Clinical Observations on Acute Otitis Media. *Proc. Roy. Soc. Med.* 37:270-274, 1944.
30. Jordan, R.: Chronic Secretory Otitis Media. *Laryngoscope* 59:1002-1015, 1949.
31. Hotchkiss, W. T.: Chronic Secretory Otitis. *South. M. J.* 41:727-732, 1948.
32. Blegvad, N. R.: Soll man den Begriff Tubaokklusion aufrechterhalten? *Monatsschr. Ohrenh.* 66:32-43, 1932.
33. Dohlman, G.: Allergiska processer i mellanörat. *Nord. med. tidskr.* 17:224-226, 1943.
34. Dohlman, G.: Till Frågan om den kroniska otitens behandling. *Nord. med. tidskr.* 20:2236-2238, 1943.
35. Koch, H.: Allergical Investigations of Chronic Otitis. *Acta Oto-laryng. Suppl.* 62:1-201, 1947.
36. Lewis, E. R.: Otitis Media and Allergy. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 38:185-188, 1929.
37. Proetz, A. W.: Allergy in the Middle and External Ear. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 40:67-76, 1931.
38. Ojala, L., and Palva, T.: Macrophages in Aural Secretions and Their Clinical Significance. *Laryngoscope* 65:670-692, 1955.
39. Derlacki, E. L.: Aural Manifestations of Allergy. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 61:179-188, 1951.
40. Suehs, O. W.: Secretory Otitis Media. *Laryngoscope* 62:998-1027, 1952.
41. Bryan, W. T. K., and Bryan, M. P.: Personal Communication.

42. Rouviere, H.: *Anatomy of the Human Lymphatic System*. Translated by M. Tobias, Ann Arbor, Edwards Bros., 1938.
43. Rüedi, L.: Mittelohrraumentwicklung und Mittelohrentzündung. *Ztschr. f. Hals-, Nasen- u. Ohrenh.* 45:175-213, 1939.
44. Rüedi, L.: Die Mittelohrraumentwicklung vom 5. Embryonalmonat bis zum 10. Lebens jahr. *Acta Oto-laryng. Suppl.* 22, 1937.
45. Beck, J.: Pathologisch-anatomische Untersuchungen über die Mastoiditis. *Passow-Schaefer Beitr.* 24:12, 1926.
46. Singer, L.: Über entzündliche Erkrankungen des Mittelohres und der pneumatischen Hohlräume des Schläfenbeines. *Ztschr. f. Hals-, Nasen-, u. Ohrenh.* 32:130-220, 1932.
47. Friedmann, I.: The Pathology of Otitis Media. *J. Clin. Path.* 9:229-236, 1956.
48. Ojala, L.: Contribution to the Physiology and Pathology of Mastoid Air Cell Formation. *Acta Oto-laryng. Suppl.* 86:1-134, 1950.
49. Friedmann, I.: The Comparative Pathology of Otitis Media - Experimental and Human. *J. Laryng. and Otol.* 69:588-601, 1955.
50. Löwy, K.: Zur Pathologie des sekretorischen Mittelohrkatarrrhs. *Monatsschr. Ohrenh.* 72:40-46, 1938.
51. Bryan, W. T. K.: The Identification and Clinical Significance of Large Phagocytes in the Exudates of Acute Otitis Media and Mastoiditis. *Laryngoscope* 63:559-580, 1953.
52. Lowry, O. H.: A Quartz Fiber Balance. *J. Biol. Chem.* 140:183-189, 1941.
53. Lowry, O. H., Rosebrough, N. J., Farr, A. L., and Randall, R. J.: Protein Measurement with the Folin Phenol Reagent. *J. Biol. Chem.* 193:265-275, 1951.
54. Kissane, J. M., and Robins, E.: The Fluorometric Determination of Deoxyribonucleic Acid in Animal Tissues with Special Reference to the Central Nervous System. *J. Biol. Chem.* In press.
55. Goa, J.: On Protein-bound Carbohydrates in Human Serum. *Scand. J. Clin. and Lab. Invest.* 7, Suppl. 22, 55 pp., 1955.
56. Gessert, C. F., Baumann, E. S., and Senturia, B. H.: Protein and Related Components in Effusions of the Middle Ear. To be published.
57. Bancroft, H.: *Introduction to Biostatistics*. New York, Hoeber-Harper, Ch. 15, 1957.

XXXV

NEUROLOGIC ASSESSMENT OF SOME
DEAF AND APHASIC CHILDREN

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Children who do not respond to sound are not necessarily deaf, nor can they all be educated by methods conventionally used with the deaf. Professional persons who work with children so handicapped, however, frequently are unable to determine whether hearing loss or some other deficiency is the basis of the symptom of inability to communicate. The educational programs to which these children are referred, therefore, may be inadequate because the programs are not geared to their particular difficulties.

A specific purpose of this study was to establish more definite criteria for differential classification of communication disorders in children. An additional purpose was to improve our understanding of the overall neurologic functioning of children with communication disorders.

SUBJECTS

The subjects of the study were 188 children with communication disorders in full-time classes at Central Institute for the Deaf. One

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hundred and fourteen had lack of sensitivity to sound or hearing loss as their only deficiency. These had been classified and were being educated as deaf children. Of the remaining 74, all being taught in a program for aphasic children, 69 were considered to be aphasic according to the criteria described by McGinnis, Kleffner and Goldstein.² Their primary disorder was the inability to express or to understand language symbols.

The aphasic children as a group were somewhat heterogeneous with respect to their disorders. In addition to variations in their receptive and expressive aphasia many of these children had additional disorders. Half of them had hearing losses as severe as some of the deaf children had. One aphasic child had emotional problems considered serious enough to warrant psychiatric treatment. Six aphasic

TABLE I
EDUCATIONAL CLASSIFICATION OF CHILDREN
AT CENTRAL INSTITUTE FOR THE DEAF

	NUMBER	PER CENT
DEAF	114	62.3
APHASIC	69	37.7

children were low enough in intelligence to be considered slightly mentally retarded by most educational standards. Nevertheless, in all of these aphasic children with additional handicaps it was the aphasic condition that was considered to be the main impediment to the development of normal verbal communication.

One of the 74 children in classes for aphasic children was attending on a probationary basis. She was eventually judged to be mentally retarded and not aphasic, and was later transferred to another kind of school. Four of the children, initially considered to have aphasic symptoms, were subsequently considered not to be aphasic on the basis of further observations in school. These five children were excluded from the study. Thus, 183 children made up the final group. One-hundred and fourteen or 62.3% were classified as deaf, and 69 or 37.7% were classified as aphasic (Table I). The final

TABLE II
ETIOLOGIC CLASSIFICATION OF CHILDREN
AT CENTRAL INSTITUTE FOR THE DEAF

	DEAF	APHASIC
UNKNOWN	59	26
PRENATAL		
Heredity		
1. Hearing loss	12	3
2. Speech disorder, CNS disorder	0	6
Rubella in first trimester of pregnancy	10	7
Other congenital complications		
1. Historical data	9	4
2. Inferred from clinical evidence of brain damage	2	5
Rh, severe jaundice shortly after birth	0	4
PERINATAL		
Complications of labor and birth	1	6
POSTNATAL		
Meningitis	9	1
Severe infection in infancy	11	1
Convulsive disorder	0	6

classification of each child was made by the school principal and supervisors on the basis of educational progress.

The data for this study came from 1) pure-tone audiograms, 2) case histories, 3) caloric tests of vestibular function, 4) neurologic examinations, 5) electroencephalograms, and 6) skull x-rays. All six observations were not made on all 183 children.

FINDINGS

Etiology. Both historical data and clinical findings were used in the evaluation of the etiologic basis of deafness and aphasia. Table II shows the etiologic classification for the children in this study. These,

of course, are not the only possible bases of communication disorders but they are the only ones which could be established with any certainty on our population. When there was a reasonable doubt about the etiologic significance of the data, the child was classified in the "unknown" category. Our judgments were conservative and hence nearly half (46.4%) of our total population is in the unknown group.

The etiologic factors are arranged in three categories: pre-, peri-, and postnatal. The prenatal factors were family histories of hearing, speech or neurologic disorders, rubella of the mother during the first trimester of pregnancy, and other congenital complications. The other congenital complications were incidents during gestation such as heavy vaginal bleeding during the early months of pregnancy; it also includes clinical evidence of brain damage or malformation without specific history of pre-, peri-, or postnatal difficulty. Jaundice resulting from Rh incompatibilities was placed in the prenatal period only because the toxic mechanism develops before the child is born.

Anoxia at birth was the principal factor in the perinatal period.

There were three principal factors in the postnatal period. The first was meningitis, and the second was severe infections, complications and treatments in infancy. This second factor can be illustrated by the case of a child who suffered severe epidemic enteritis at the age of three days. She ran a high fever, became dehydrated, and was treated with large doses of streptomycin and other antibiotics. Three other children in the nursery died from the same infection. Convulsive disorder without apparent precipitating cause was considered of etiologic significance in six children. Seizures were recurrent in five of them. Two children lost the speech and understanding of speech they had,¹ and in four children there was no development of verbal communication. Ten other children (five deaf and five aphasic) also had seizures, five of them with an episode of acute meningitis, and five with other acute illnesses (infection, jaundice, uremia, etc.). None of the deaf children had recurrent convulsive seizures.

Etiologic factors are related to some extent to the nature of communication disorders. For instance, of the 22 children who had meningitis or other severe infections in infancy more than 90% were deaf. By contrast, four children with histories of Rh incompatibility, six of the seven children with histories of difficult birth, and all of the six with convulsive disorders were aphasic.

TABLE III
ETIOLOGIC CLASSIFICATION OF CHILDREN
AT CENTRAL INSTITUTE FOR THE DEAF
(REVISED AND CONDENSED CATEGORIES)

	DEAF	APHASIC
Meningitis		
Severe Infection in Infancy	32	5
Family History of Hearing Loss		
Maternal Rubella	19	11
Complication During Pregnancy		
Rh		
Complication of Labor and Birth		
Convulsive Disorder	3	27
Congenital Brain Abnormality		
Family History of Speech or Neurologic Disorder		

Some etiologic groupings were developed to separate the deaf from the aphasic children as clearly as possible (Table III). In the first group are the postnatal factors of meningitis and other severe infantile infection, and family history of hearing loss. The second group includes maternal rubella and other complications during pregnancy. In the third group are the factors of Rh incompatibility, complication of labor and birth, convulsive disorder, congenital brain abnormality, and family history of speech or neurologic disorder.

The first group is heavily weighted with deaf children. Meningitis, in particular, is commonly believed to damage the auditory nerve. The third group is weighted with aphasic children, and factors in this category are commonly associated with some impairment of the central nervous system. The middle group is divided in approximately the same proportion as the over-all proportion of deaf and aphasic children in our total population. Many different kinds of impairments, nonauditory as well as auditory, have been attributed to maternal rubella and to such incidents as heavy vaginal bleeding during pregnancy.

Audiometry. Auditory thresholds were determined by conventional behavioral audiometry. We chose the audiogram as the best

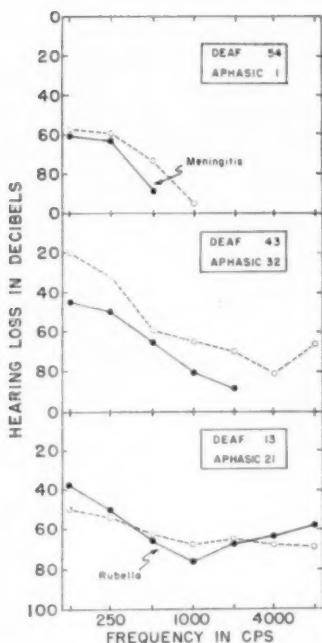


Fig. 1.—Each curve represents the 50th percentile of the distribution of audiograms with similar configurations but with different degrees of hearing loss. The number of deaf and aphasic children whose audiograms are classified in either of the two patterns in each of the three parts of the figure are shown in the box in the upper right section of each part.

single measure of auditory sensitivity because it describes the distribution as well as the severity of the hearing loss. All of the audiograms were classified in one of the patterns shown in Figure 1.

Nine normal audiograms, all of aphasic children, are not shown in Figure 1. The aphasic children in general tend to have only moderate hearing losses and, on the average, relatively flat audiograms; the deaf children, more severe losses and sloping audiograms.

We find distinctive audiometric patterns associated with some etiologic factors. Maternal rubella seems to produce the most char-

acteristic audiogram, a U-shaped or trough-shaped curve with its lowest point, about 75 db, at 1000 cps. Meningitis commonly produces the residual type audiogram with responses only for low frequencies and only at the limits of the audiometer. In general, there is a predominance of histories of meningitis and other severe infantile infections among the children with severe hearing losses. On the other hand, the majority of children with normal hearing or with flat audiograms have in their histories some incident suggesting impairment of the central nervous system.

TABLE IV
VESTIBULAR RESPONSES OF DEAF AND APHASIC CHILDREN
AT CENTRAL INSTITUTE FOR THE DEAF

	TOTAL POPULATION				RESTRICTED POPULATION*			
	DEAF		APHASIC		DEAF		APHASIC	
	N	%	N	%	N	%	N	%
Normal	55	51.4	21	36.8	50	56.8	21	38.2
Depressed	25	23.4	25	43.9	24	27.2	25	45.5
No Response	27	25.2	11	19.3	14	15.9	9	16.3

* Excluding children with history of meningitis or other severe infantile infection.

Vestibular Tests. Vestibular function was tested by a modified form of the Hallpike-Cawthorne test. (The vestibular tests were performed by Dr. Benjamin Rosenblüt, World Health Organization Fellow, Hospital of San Juan de Dios, Santiago, Chile.) Because we originally intended only to screen out those who gave no responses, we used a strong stimulus, water at 18° C for 40 seconds. Several aspects of the resulting nystagmus were recorded, but duration from the beginning of the flow of water was chosen as the main criterion of responsiveness. Table IV gives the results of our findings. We classified the responses as normal, depressed, or absent (i.e., no response). Our norms were based on responses from 16 children with normal hearing.

Contrary to our expectations, a larger proportion of the deaf children, 51.4%, than of the aphasic children, 36.8%, gave normal

responses (Table IV). When the table is revised to exclude those children with histories of meningitis or other severe infantile infections there is proportionally an even greater prevalence of normal responses among the deaf children, 56.8%, than among the aphasic children, 38.2%. The aphasic group have a disproportionately large percentage, 45.6%, of children with depressed vestibular responses, compared to 27.2% for the deaf children.

Neurologic Examination. This was a standard initial physical examination routinely performed by a neurologist. The most obvious abnormalities noted were of the motor system. These were classed as either minor or major abnormalities. The minor abnormalities were such things as nystagmus or strabismus. The major abnormalities were mostly evidences of pyramidal or extrapyramidal impairment; 23.2% of the aphasic children showed major abnormalities (Table V) compared with less than 4% of the deaf children. About

TABLE V
MAJOR MOTOR ABNORMALITIES AMONG DEAF
AND APHASIC CHILDREN
AT CENTRAL INSTITUTE FOR THE DEAF

	NUMBER	PER CENT
DEAF	4	3.5
APHASIC	16	23.2

25% of both deaf and aphasic children also showed minor neurologic abnormalities.

We have applied the label "obtuseness" to an additional observation. This consisted of a frustrating incapacity of the child to comprehend a nonverbal perceptive task (e.g., to touch a part or parts of the body touched by the examiner, or to signal the visual awareness of a target light). This was not a question of lack of co-operation or poor behavior during the examination. Obtuseness was noted in 16 of the 69 aphasic children (23.2%) and in only 2 of the 114 deaf children (1.8%) (Table VI). Obtuseness most likely parallels the behavior identified by the educational staff as aphasic.

TABLE VI
"OBTUSENESS" NOTED DURING NEUROLOGIC
EXAMINATION

	NUMBER	PER CENT
DEAF	2	1.8
APHASIC	16	23.2

Electroencephalography. The electroencephalograms were standard waking records, including activation by hyperventilation. The records were read conservatively by one electroencephalographer and reviewed by another who was furnished no information except the age of the child. Any records judged borderline were classified as normal. About 40% of both deaf and aphasic children had abnormal EEGs. The abnormalities ranged from abnormal mixtures of slow and fast activity to focal spikes. There were more focal abnormalities in the EEGs of the aphasic children (14.5%) than in those of the deaf children (6.1%). The unexpected finding was the presence of so many abnormal EEGs among the deaf.

Skull X-rays. X-ray abnormalities were observed in seven of the 157 children who had skull x-rays. In two, occipital flattening of the cranial vault was observed, and in two others there was parietal asymmetry. All four of these children were aphasic. Evidence of chronic mastoiditis was seen in three children, two aphasic and one deaf. Thus, it appears that anomalous development of the cranial vault may correlate with the educational classification of aphasia.

CONCLUSIONS

Etiologic background appears to contribute most to the task of differentiation. Meningitis and other severe infantile infections, and family history of hearing loss support the classification of deafness. Jaundice resulting from Rh incompatibility, anoxia at birth, convulsive disorder, congenital brain abnormality, and family history of speech or neurologic disorder support the classification of aphasia. Maternal rubella during the first trimester of pregnancy and other

TABLE VII
DIFFERENTIATING CHARACTERISTICS OF DEAF AND APHASIC CHILDREN

	MOST USEFUL			LEAST USEFUL		
	ETIOLOGY	AUDITORY	MOTOR SYSTEM	VESTIBULAR	EEG	SKULL X-RAYS
DEAF	meningitis	sloping audiogram	no major abnormality	normal, except for children with meningitis, etc.	-----	-----
	severe infantile infection					
	family history of hearing loss					
DEAF or APHASIC	maternal rubella	sloping audiogram	minor abnormality	normal, depressed or no response (with sloping audiogram and moderately severe hearing loss)	normal or dysrhythmic	normal
	complication during pregnancy	moderately severe hearing loss				
APHASIC	Rh	normal hearing or flat audiogram with moderate hearing loss	major abnormality	depressed (with normal hearing or moderate hearing loss)	focal abnormality	anomaly of cranial vault
	complication of labor and birth					
	convulsive disorder					
APHASIC	congenital brain abnormality					
	family history of speech or neurologic disorder					

complications during pregnancy may result in deafness, or in aphasia and deafness.

Audiometric studies are also helpful in differentiation but audiograms are not usually easy to obtain on very young children, at least with present behavioral techniques. The sharply sloping audiogram and severe hearing loss is characteristic of deaf children; normal sensitivity or a moderate hearing loss for all frequencies is characteristic of aphasic children. However, sloping audiograms with moderate to severe hearing losses occur frequently in both groups.

Major motor abnormalities appear more frequently in aphasic children than in deaf children. Minor motor abnormalities occur in about one-fourth of both deaf and aphasic children and thus are not differentiating. Obtuseness during neurologic examination is much more frequent among the aphasic than among the deaf children.

Normal vestibular responses are more likely to occur in deaf rather than aphasic children with the exception that most of the children with histories of meningitis or other severe infantile infections usually give no vestibular responses. Depressed vestibular responses in the presence of moderate or normal auditory sensitivity is more characteristic of aphasic children.

Electroencephalograms are not very differentiating despite the rather large percentage of abnormal EEGs. Focal abnormalities are more frequent among aphasic children.

Anomalies of the cranial vault as seen in skull x-rays are more likely to occur in aphasic than in deaf children.

None of the findings provide unequivocal criteria for differentiation among very young deaf and aphasic children. They do, however, provide combinations of essentially neurologic observations that can increase the accuracy of differentiation in advance of actual educational experience.

SUMMARY

Six different measures or evaluations were made on 183 deaf and aphasic children at Central Institute for the Deaf. The purpose of

the study was to establish more definite criteria for differential classification of communication disorders in children in advance of actual educational experience. The validating criterion for deafness and aphasia was the observation of progress made by each child in his educational program. The findings are summarized in Table VII.

ACKNOWLEDGMENTS—We thank Dr. James O'Leary and Dr. William Seaman of the Washington University School of Medicine for their help in obtaining and interpreting the EEG records and x-ray films. We also thank the following persons at Central Institute for the Deaf: Dr. Hallowell Davis and Dr. S. Richard Silverman for their support and guidance during the conduct of the study, and for their criticisms of our paper; Dr. Robert C. Bilger for his invaluable suggestions and help in the processing of the data; and the teachers and parents of the pupils for their generous co-operation.

REFERENCES

1. Landau, W. M., and Kleffner, F. R.: Syndrome of Acquired Aphasia with Convulsive Disorder in Children. *Neurology* 7:523-530, 1957.
2. McGinnis, M. A., Kleffner, F. R., and Goldstein, R.: Teaching Aphasic Children. *Volta Review* 58:239-244, 1956.

Scientific Papers of the American Laryngological Association

XXXVI

GROUND SUBSTANCE IN THE NOSE IN HEALTH AND INFECTION

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AND

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(Both by invitation)

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How does an infection spread in the nasal mucosa? What initiates the common cold or any other infection? Are there specific organisms involved? Why does one member of a family not develop a nasal infection when all the other members of the same family do? Is it entirely a matter of individual systemic resistance or are there some specific differences locally in the nose? Who has not been interested in these questions? The answers could help to provide methods of preventing nasal infections.

It was in 1928 that Duran-Reynals¹ published the first observations on a spreading factor, which increased connective tissue permeability. Ten years later Chain and Duthie^{2,3} identified this spreading factor as hyaluronidase, capable of depolymerizing, thus decreasing the viscosity and thereby increasing the permeability of the intercellular mucin-like substance.

With this introduction, considerable study and research was devoted to connective tissue, its composition and its function in the organism. As pointed out by Goldsmith,⁴ Duran-Reynals' observations and penetrating inferences catalyzed a tremendous quantity of research in the three decades following his original report.⁵⁻⁸ Organic

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and enzyme chemists, microscopists, oncologists, endocrinologists, gynecologists and pharmacologists were among those who made important contributions. Angevine¹⁰ points out that connective tissue comprises a number of tissues that arise from mesoderm and are concerned with the formation and maintenance of bodily structure. In outlining areas upon which all participants agreed, for the Fourth Conference on Connective Tissues, Ragan⁹ listed the divisions of connective tissue as follows:

Normal structure

- I. Cellular
 - a. Fibroblast
 - b. Macrophage
 - c. Mast cell
- II. Fibrillar
 - a. Collagen
 - b. Reticulin
 - c. Elastic fibers
- III. Interfibrillar material "ground substance"
(The particular cell which secretes the ground substance is a matter of dispute; fibroblast [Gersh], mast cell [Asboe-Hansen]).
 - a. Mucopolysaccharide component
 - b. Protein components - unknown

Abnormal states

- I. Fibrinoid
- II. Collagen and ground substance in disease
- III. Responses to trauma
 - a. Local
 - b. Antibodies

The tremendous importance of the ground substance in resistance to infection was established in a fine, scholarly, thorough study by Duran-Reynals¹¹ in 1942. He demonstrated that only certain organisms contain spreading factors (hyaluronidase). In staphylococcal organisms the spreading factor is particularly well demonstrated.

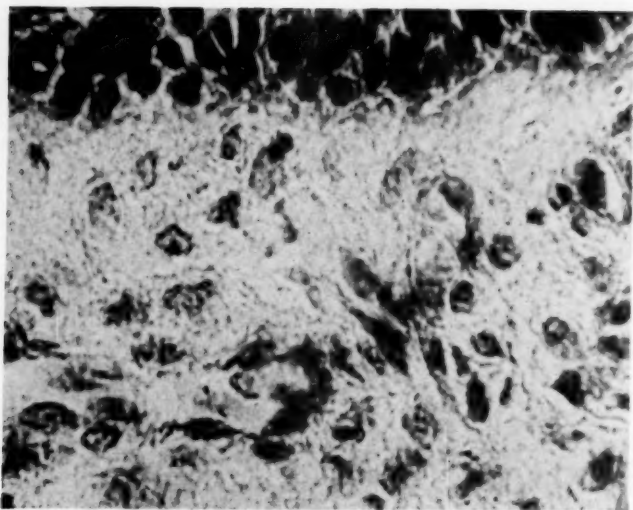


Fig. 1.—Photomicrograph of a normal turbinate; black and white reproduction of a Schiff-stained section.

Various strains of staphylococci have varying amounts of the spreading factor. In streptococcal infections the process is somewhat more complicated as there are some strains which have hyaluronic acid (ground substance) as well as some which have hyaluronidase (spreading factor). Hobby et al¹² have felt that there were spreading factors other than hyaluronidase in some streptococcal strains. Our observations tend to confirm this finding. Duran-Reynals¹³ has shown that the invasiveness of a strain of streptococcus was largely determined by the amount of spreading factor. It should be indicated here that there is a difference between invasiveness and virulence. Virulence depends on a general or local susceptibility in the host and toxogenicity of the bacteria. Invasiveness merely indicates the spreading ability. As stated by Duran-Reynals,¹³ "the experiments reported here seem to justify the conclusion that the invasion of the skin by staphylococcus depends on the presence in the micro-organism of a substance which markedly increases the permeability of the tissue thus rendering invasion more easy." Several

authors have demonstrated spreading factors in pneumococci.¹⁴⁻¹⁶ Similar studies have yielded spreading factors in other organisms.^{14,17,18}

As a further corollary of the problem of the manner of spread of infection in the nose, Hanger¹⁹ as far back as 1931 demonstrated that filtrates of nasal secretions from normal individuals increased spread of leptospirium infection injected in the skin of rabbits. Secretions from patients with acute colds accelerated the spread more than secretions taken during the normal period.

Thus far then, we can accept the presence of an amorphous ground substance in connective tissue which contains a viscid acidic substance²⁰ identified by Meyer and Palmer²¹ in 1934 as an acid mucopolysaccharide and named by them hyaluronic acid. This substance in its polymerized state is resistant to the spreading of the organisms of infection. Some six mucopolysaccharides have been identified in different tissues.²² Hyaluronidase or similar spreading factor which has been found in some bacteria and not in others is capable of depolymerizing the hyaluronic acid and thus increasing its permeability. With these facts in mind it seemed to me that the occurrence of nasal infection might be the result of infection with the particular organisms containing spreading factor or the result of areas of overgrowth of these organisms initiating spread through the so-called basement membrane of the ground substance of the nose and thereby causing dissemination of infection.

It is known that a hyaluronidase system is dependent on a certain pH for optimum activity and perhaps alteration of pH is a factor in initiating the process. Hale²³ showed that hyaluronidase derived from either group A or group C streptococci is rapidly inactivated at pH 4.6 but is relatively stable at pH 7.0. Meyer and his co-workers^{16,24} showed that the peak of activity of hyaluronidase of cl. *Welchii* and streptococcus and pneumococcus origin was between 5.2 and 6.5. There was no work uncovered on the effect of varying pH on hyaluronidase inhibitors and most workers kept their pH for inhibitor studies between 6.0 and 7.5.

In 1950, I thought I might perhaps be able to demonstrate an increase in hyaluronidase-active organisms in the nose in infection. This required the assay of minute amounts of hyaluronidase. The

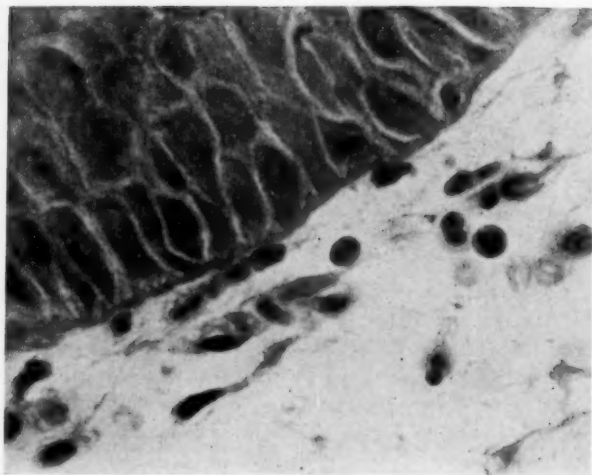


Fig. 2.—Photomicrograph showing depolymerization of ground substance in an infected nasal polyp.

turbidimetric method of hyaluronidase activity determination could not be used with the amounts conceivably altered in nasal infection. Quinn²⁵ at the University of Wisconsin in June 1950 advised me of a method devised by C. V. Seastone there, for determining the presence of minute amounts of hyaluronidase in bacteria. Unfortunately the method was not quantitative.

Biochemical analysis of nasal tissues would not furnish specific information as to the site and character of the changes in the nose in infection.

The project was laid aside until about a year ago when Dr. Weisskopf of our Department became interested in the histochemical demonstration of changes in nasal ground substance in patients with hormonal problems. This seemed an excellent means for studying the changes in ground substance in the nose in infection.

A careful survey of the literature in English showed no work on the structure of the ground substance in the nose. There were a few papers in the Italian literature and fewer in the German.

While it was felt that a true picture of the physiological function of ground substances in the nose²⁶ depended on a correlation of chemical studies, histochemistry, physiochemistry, metabolism studies, functional and pathological studies, and while the lack of specificity of many of the histochemical techniques was recognized, it was felt that since this would be a comparative study of identical tissue, much information could be gained.

One of us (H. B.) undertook a thorough review of various histochemical techniques to determine their specificity in demonstrating mucopolysaccharides of the hyaluronic acid type of which nasal ground substance was presumed to be composed. Our conclusions were much like those of Davies.²⁷ The classical ground substance stains when tested against hyaluronic acid are often not specific. Ground substance in the nose probably has other acid mucopolysaccharides than hyaluronic acid and in staining nasal tissue the ground substance can certainly be demonstrated.

In five patients with normal nasal mucosa a biopsy of the turbinate was taken under topical anesthesia just prior to performing a submucous resection. To reduce the possible alteration of ground substance by the shrinking of the mucosa, only pontocaine was used for local anesthesia of the turbinate. The same type of anesthesia was employed throughout the study. Three patients with a unilateral purulent maxillary sinusitis had biopsies of their turbinates just prior to irrigation of purulent material from the maxillary sinus. In addition, unilateral nasal polyps of the infection variety were removed and included in the study. The tissues were fixed in 10 per cent neutral formalin, embedded in paraffin (52-54° Tissue Mat). The material was serially sectioned at 5-7 micra. The serial sections were treated as follows:

One section was treated with testicular enzyme (hyaluronidase), one with bacterial enzyme (hyaluronidase), and the third section was not treated. In the staining study, azure-eosin: HCL-Orange G-Methyl Blue; several Alcian Blues, Steedman's and Lison's included, were tried and discarded as not being sufficiently specific. Harris' hematoxylin and eosin, the standard stain for pathological tissues, was used primarily for demonstration of how little could be seen of the important connective tissue ground substance, for comparison with the less well known stains and for routine histology and cytology.

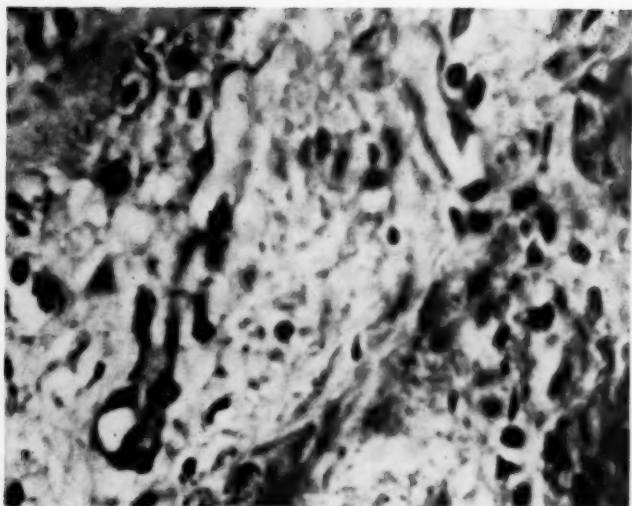


Fig. 3.—Photomicrograph of a normal turbinate showing ground substance in the right lower corner; colloidal iron stain.

Kramer and Windrum Azure A and Gomori's toluidine blue were compared for metachromasia and toluidine blue was selected as one of the stains to be used. It demonstrated metachromasia of the ground substance and particularly of the mast cells. Periodic acid-Schiff was selected since it is a commonly used stain for ground substance demonstration. The fourth and last stain of the group was the Rinehart and Abul-Haj²⁸ modification of the Hale Colloidal Iron technique. Here again the original specificity indicated by Rinehart has been disputed by other investigators but with the combination of stains as done in this project, it was felt considerable information could be obtained about changes in ground substance.

It must be acknowledged that no one yet knows the composition of nasal ground substance. It is probably mainly acid mucopolysaccharide or hyaluronic acid. To show the complexity of this ground substance the molecular weight is estimated at 500,000 which is probably much too low as has been shown by Meyer.⁴

In addition to turbinate biopsies in patients with normal mucosa and those with infection a series of embryo turbinates were studied with the same four stains.

Study of the turbinates from the normal individuals shows ground substance beautifully. There is demonstrated a concentration of the material just under the ciliated epithelium which probably is, in effect, the basement membrane. In patients with infection and in the infected nasal polyp the changes of depolymerization can be seen definitely. The basement membrane area of concentration of ground substance has disappeared and more metachromatic substance can be seen on the surface of the ciliated epithelium. This may represent a defense mechanism. In the turbinates treated with bacterial hyaluronidase similar changes to those in infection may be seen but to a lesser degree than in the more potent fresh testicular hyaluronidase.

Both of these changes are to a lesser degree than the change in infection. It is not entirely clear that this is a matter of degree alone. There is a strong suggestion that other enzyme systems may be involved in the depolymerization and fluidifying process of the ground substance with its subsequently increased permeability.

That others have been thinking and working along similar lines can be seen in the articles of Lisanti,²⁹ Aisenberg,³⁰ and Schultz-Haudt.³¹ Lisanti demonstrated that in 20 patients with periodontal disease ranging from gingivitis to periodontoclasia, all had measurable hyaluronidase activity in the saliva. Of the remaining patients in the studied group of 64, those who had no periodontal disease had no hyaluronidase activity. He felt hyaluronidase activity of the saliva or its cocci might be a factor in the initiation or progress of dental caries. Aisenberg and Aisenberg³⁰ were able to show by injection of hyaluronidase into the interdental papillae of monkeys that the changes, microscopically, were the same as those in humans affected by periodontal disease. They were of the opinion that those bacteria which secreted hyaluronidase were an etiological factor in the production of periodontal disease. Schultz-Haudt et al³¹ in a study of the production of gingival inflammation demonstrated a spreading factor in cell free extracts of gingival debris. They treated human gingiva with hyaluronidase and noted marked connective tissue changes in biopsies taken after one and one-half days' exposure. Their con-

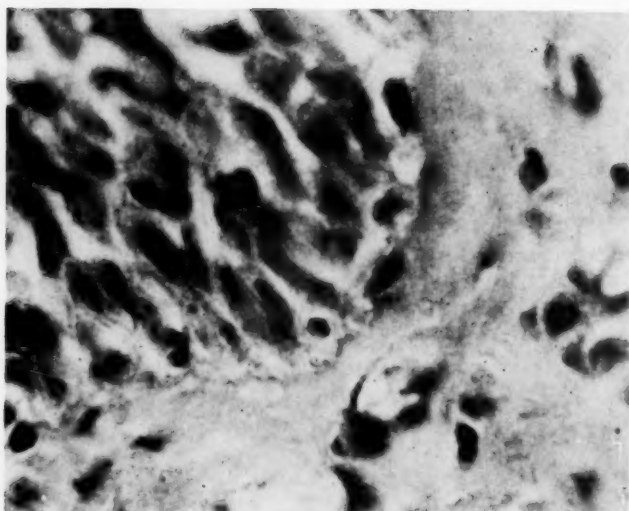


Fig. 4.—Photomicrograph of infected turbinate showing depolymerized ground substance with colloidal iron stain.

clusions are most interesting. "It may be concluded that the initial effects of hyaluronidase on epithelium are an increase in polysaccharide containing materials and prolonged action which is capable of altering the intercellular substance in the gingival epithelium thereby permitting passage of destructive agents to the underlying connective tissues. . . . On the basis of these findings, it is suggested that the various hyaluronidase-like products of gingival bacteria may produce similar changes in the gingiva and thus contribute to gingivitis."

CONCLUSION

From a comparative multiple stain technique of serially sectioned biopsies of turbinates from normal individuals and from patients with infection, it can be demonstrated that a depolymerization and thus increased permeability, of ground substance, occurs. The change is similar to that produced in the normal tissues when treated with hyaluronidase but also suggests that other enzyme systems are in

operation. The relationship of spreading factors to ground substance is probably a dynamic one varying with functional conditions. Certain bacteria have been demonstrated by other workers to possess spreading factors. Overgrowth of these organisms in isolated areas of nasal mucosa could initiate spread of infection. Since hyaluronidase is most active in the range of normal, slightly acidic nasal pH the accepted concept of alteration of nasal pH to the alkaline side in nasal infection may be a defense mechanism and therefore the effect of the early stage of infection rather than the cause. Hyaluronidase inhibitors or substances which would help maintain the normal gel state of nasal ground substance should, at least theoretically, halt the spread of nasal infection.

2000 VAN NESS AVE.

REFERENCES

1. Duran-Reynals, F.: Exaltation de l'activite du virus vaccinal par les extraits des certains organes. *Compt. rend. soc. biol.* 99:6, 1928.
2. Chain, E., and Duthie, E. S.: *Nature* 144:977 (Dec. 9) 1939.
3. *Ibid.*: *Brit. Jour. Exper. Path.* 21:324 (Dec.) 1940.
4. Goldsmith, E. D.: Ground Substance of the Mesenchyme and Hyaluronidase. *Ann. N. Y. Acad. Sci.* 52:7:945 (May 31) 1950.
5. Persson, B. H.: Studies on Connective Tissue Ground Substance. *Act. Soc. Med. Upsal.* 58:2, 1953.
6. Asboe-Hansen, G.: Connective Tissue in Health and Disease. *Philosoph. Libr., N. Y.*, 1957.
7. Ciba Foundation Symposium on Chemistry and Biology of Mucopolysaccharides. J. & A. Churchill, Ltd., London, and Little, Brown & Co., Boston, 1958.
8. Anderson, O.: Eine methode zum Nachweis des Vakzinevirus in den Organen vakzinierter Kaninchen. *Z. Immun.* 90:105, 1937.
9. Ragan, Charles: Transactions of the Fourth Conference on Connective Tissues. Josiah Macy, Jr. Foundation, p. 16, 1953.
10. Angevine, D. M.: Transactions of the First Conference on Connective Tissues. Josiah Macy, Jr. Foundation, N. Y., p. 13, 1950.
11. Duran-Reynals, F.: Tissue Permeability and the Spreading Factors in Infection. *Bact. Rev.* 6:197, 1942.
12. Hobby, G. L., Dawson, M. H., Meyer, K., and Chaffee, E.: The Relationship Between Spreading Factor and Hyaluronidase. *Jour. Exp. Med.* 73:109, 1941.
13. Duran-Reynals, F.: Studies on a Certain Spreading Factor Existing in Bacteria and its Significance for Bacterial Invasiveness. *Jour. Exp. Med.* 58:161, 1933.

14. McClean, D.: A Factor in Culture Filtrates of Certain Pathogenic Bacteria Which Increases the Permeability of the Tissues. *Jour. Path. Bact.* 42:477, 1936.
15. McClean, D., and Hale, C. W.: Studies on Diffusing Factors. *Biochem. Jour.* 35:159, 1941.
16. Meyer, K., Chaffee, E., Hobby, G. L., and Dawson, M. H.: Hyaluronidases of Bacterial and Animal Origin. *Jour. Exp. Med.* 73:309, 1941.
17. McClean, D.: "Substance B" of Diphtheria Toxin and Diffusing Factor. *Lancet* 240:595, 1941.
18. O'Meara, R.A.Q.C.: Diphtheria and the Composition of its Toxin in Relation to the Severity of Diphtheria. *J. Path. Bact.* 51:317, 1940.
19. Hanger, F. M.: Influence of Secretions of the Upper Respiratory Tract on Tissue Resistance. *Proc. Soc. Exp. Biol. Med.* 29:285, 1931.
20. Stacey, M.: The Chemistry of Mucopolysaccharides and Mucoproteins, Advances in Carbohydrate Chemistry. Academic Press Inc. 2:161, 1946.
21. Meyer, K., and Palmer, J. W.: The Polysaccharide of the Vitreous Humor. *Jour. Biol. Chem.* 107:629, 1934.
22. Meyer, K., Davidson, E., Linker, A., and Hoffman, P.: The Acid Mucopolysaccharides of Connective Tissue. *Biochem. Biophys. Acta* 21:506, 1956.
23. Hale, C. W.: The Influence of Some Environmental Conditions on the Activities of Hyaluronidase. *Biochem. Jour.* 38:368, 1944.
24. Meyer, K., Hobby, G. L., Chaffee, E., Dawson, M. H.: The Hydrolysis of Hyaluronic Acid by Bacterial Enzymes. *Jour. Exp. Med.* 71:137, 1940.
25. Quinn, Robert W.: Univ. of Wisconsin, Madison, Wis., Personal Communication.
26. Moore, R. D., and Schoenberg, M. D.: Studies on Connective Tissue. *A.M.A. Arch. Path.* 64:39 (July) 1957.
27. Davies, D. V.: Specificity of Staining Methods for Mucopolysaccharides of the Hyaluronic Acid Type. *Stain Tech.* 27:65, 1952.
28. Rinehart, J. F., and Abul-Haj, S. K.: An Improved Method for Histologic Demonstration of Acid Mucopolysaccharide in Tissues. *A.M.A. Arch. Path.* 52:189, 1951.
29. Lisanti, V. F.: Hyaluronidase Activity in Human Saliva. *Jour. Dent. Res.* 29:3:392 (June) 1950.
30. Aisenberg, M. S., and Aisenberg, A. D.: Hyaluronidase in Periodontal Disease. *Oral Surg. Oral Med., Oral Path.* 4:3:317 (Mar.) 1951.
31. Schultz-Hautd, S., Dewar, M., Bibby, B. G.: Effects of Hyaluronidase on Human Gingival Epithelium. *Science* 117:653 (June 12) 1953.

XXXVII

VOCAL REST IN LARYNGEAL DISEASE

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Observation over a period of years indicates that the application of silence or voice rest is not well understood; very little has been written on this subject. It is significant that only one article could be found in the literature which deals specifically with voice rest.¹ Silence is used frequently for prolonged periods without benefit to the patient. There appear to be two reasons for this. First, many cases of hoarseness are diagnosed by inference; and second, there seems to be a lack of understanding of the tissue changes involved when voice rest is prescribed. Certain conditions respond to voice rest, others do not. Acute and chronic inflammations, certain cases of specific infection such as tuberculosis, ulcerations, and small tumors, including well-localized cordal cancer, in both their pre-operative and post-operative phases, require this form of therapy.

Essential to the understanding of the value of silence and its application is a knowledge of the tissue changes involved. Consideration must be given also to the location and extent of the lesion. In all inflammations there are two early changes which can be detected by clinical study. These are edema and swelling. Later changes depend principally upon the deposit of fibroblasts and the formation of fibrous tissue, so that the early soft edematous lesion frequently becomes a firm one. Edema and swelling frequently precede and usually coexist with vocal cord tumors. The presence of fibrous tissue bespeaks permanency. For this reason it does not matter whether the patient is forbidden to use his larynx for a day, a month or a year; no benefit will come of it, because fibrous tissue is not reversible. The only changes which are reversible are the early edema and swelling. Since this is so, the use of silence or voice rest can only be effective when applied at this stage.

It might be well to consider two conditions, the management of which appears to be controversial. These are the vocal nodule and

the contact ulcer. Many authors advocate three months or more of vocal rest for the elimination of a vocal nodule. Unfortunately, the results of this prolonged regime are rarely satisfactory. It must be admitted that three months or more of silence is not very desirable from the standpoint of the patient, if the results are questionable, and removal of the nodule will give him a good voice within a few days.

A vocal nodule in its early stage is a purely inflammatory lesion consisting principally of edema. It is at this time that 24 hours of vocal rest will cause the lesion to disappear. Permanency of the nodule is caused by the introduction of fibroblast. The best thing to do for the patient with a vocal nodule is to remove it with suitable instruments. This is best accomplished by the use of indirect laryngoscopy. When removal is performed by indirect laryngoscopy, the possibility of injuring the vocal cords is greatly reduced. Some have been apologetic for attempting the removal of so small a mass. This is difficult to understand.

In the management of contact ulcer, the use of vocal rest will depend upon the tissue changes present. Here the laryngologist must evaluate the nature of the lesion; he must decide whether it is active, quiescent, or healing. If it is active, a period of strict silence should be insisted upon. If it is inactive or healing, however, the patient should be allowed to speak without emphasis or exertion. If he is not able to do this, it becomes necessary to insist upon complete vocal rest. Healing seems to be enhanced by using the voice at this stage. Voice therapy has proven of value in this condition.

Another condition worthy of special consideration is the localized edema which occurs on a vocal cord as the earliest sign of reaction to excessive smoking. The edema disappears within 12 to 24 hours if smoking is discontinued and the voice placed at rest² (Fig. 1).

Vocal rest is important in the pre-operative as well as the post-operative management of tumors. In the pre-operative phase it is important to recognize that practically all lesions of the vocal cord are surrounded by a zone of edema; occasionally true inflammation exists. It is important, therefore, that the vocal cord be rested 24 hours to eliminate the edema; if inflammation is present, the period of rest is longer. Healing and a return to normal are much more rapid when this indication is recognized and employed. Therefore,

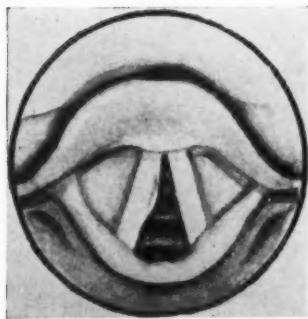


Fig. 1.—Early edema in smoker's larynx; reversible by vocal rest.
(Reprinted from the ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY
59:541 (June) 1950)

all localized tumors of the larynx should be given the benefit of voice rest immediately before operation.

The most important application of silence or voice rest is related to the period after removal of a vocal cord tumor. In most cases the actual lesion caused by removal is small, but reaction of the adjacent tissues or the entire cord to trauma may cause the lesion to be quite extensive.

The success of the operation for removal of a vocal cord tumor depends upon the complete and clean-cut removal of the growth, and restoration of the voice to normal. Ideal removal of such a neoplasm is that which is attended with no visible injury to the surrounding structures. This is possible when a lesion is discrete, such as a polyp, a nodule, or a localized cancer or papilloma. In such cases the patient, if intelligent, may be instructed to speak softly and use his voice sparingly. Even in those cases where there is denudation of the mucous membrane closely adjacent to the site of the tumor, cautious use of the voice will do no harm. By cautious use of the voice is meant the absence of strain such as accompanies loud or emphatic speaking or shouting.

The vocal cord is a very vulnerable structure; it is extremely sensitive to injury. Subepithelial hemorrhage and edema occur readily

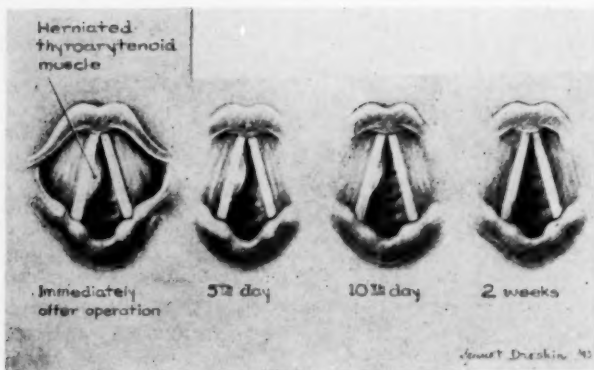


Fig. 2.—Herniation of thyro-arytenoid muscle following stripping of vocal cord. Restoration to normal in two weeks. (Reprinted from *Arch. of Otolaryng.* 39:250 (Mar.) 1944)

when a cord is traumatized. Also, it is very easy to strip the mucous membrane of a vocal cord especially in the area adjacent to a firmly attached neoplasm. Postoperative lesions range from a simple, minute removal of epithelium to extensive sacrifice not only of the epithelium, but also of the subepithelial tissues and part of the thyro-arytenoid ligament. The vocal cord may be so injured as to later present a continuous band of granulation tissue. Vocal rest is of little value in the presence of a benign granulomatous lesion of the vocal cord or a loss of its substance. Loss of structure of the vocal cord is overcome by a straightening-out process, which can best be accomplished by using the larynx. It has been noted that epithelization of the vocal band will continue regardless of whether or not the voice is used, provided it is used with caution.

An understanding of the time factor in epithelization of the vocal cord was furnished by an interesting experience some years ago. During the removal of a vascular polyp, the mucous membrane was denuded along the entire free margin of the vocal cord. This was followed immediately by an extensive herniation of the thyro-arytenoid muscle. The larynx was examined daily. At the end of 14 days epithelization was complete and the larynx normal³ (Fig. 2).

Following removal of a tumor, the period of vocal rest should be as short as possible. This is to avoid psychological trauma which frequently accompanies a regime of prolonged complete silence. Frequently, fear that one might not be able to speak again after such a period of silence is real. It is therefore important to explain to the patient that resting the voice is necessary to overcome the effects of the operation, and to return the voice to normal.

It is not necessary to extend the period of silence beyond the time when the process of healing and resolution are well advanced. In some cases return to normal may be delayed a few days, but the strain, annoyance, and frustration of maintaining silence will be eliminated. If the patient will speak softly and without strain once the healing has progressed to a point where ulceration and edema are no longer present and thickening of the cord has subsided, no harm will be done. There are a few who believe that the healing process is accelerated by such postoperative management. Experience has shown that cautious and intelligent use of the voice at this stage hastens rather than retards recovery.

This paper deals with the application of voice rest which has not been too well-defined in the past. A comprehension of the tissues changes which occur before and after removal of vocal cord tumors will furnish a better understanding of the application of this form of therapy.

416 NORTH BEDFORD DRIVE

REFERENCES

1. Cantor, J. J.: A Method of Resting the Vocal Cords. *Laryngoscope* 67:157 (Feb.) 1957.
2. Myerson, M. C.: Smoker's Larynx. *ANNALS OF OTOLGY, RHINOLOGY AND LARYNGOLOGY* 59:2:541 (June) 1950.
3. Myerson, M. C.: Vascular Polyp of the Vocal Cord. *A.M.A. Arch. Otolaryng.* 39:250-258 (Mar.) 1944.

XXXVIII

THE MANAGEMENT OF CHRONIC LARYNGEAL STENOSIS

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Chronic laryngeal stenosis is usually associated with a distressing degree of disability. Physical impairment ranges from respiratory effort, stridor, and even chronic anoxemia to actual acute obstruction which may have to be relieved mechanically by a tracheotomy. Mentally, the disability is aggravated by the annoyance of the tracheotomy tube and the psychic stress of an abnormal, peculiar voice which is a constant source of embarrassment. This continues far beyond the first contact with others; children so afflicted are subject to teasing and insults from their playmates and adults have difficulty not only socially, but in finding or keeping steady employment.

Rehabilitation of the patient with chronic laryngeal stenosis has three requisites: first, re-establishment of the normal airway and closure of the tracheostomy; second, the restoration of as serviceable a voice as possible, and aiding the patient in overcoming the embarrassment of what is frequently exceedingly abnormal speech; third, to provide a larynx that will close satisfactorily to prevent the aspiration of food and fluids during deglutition.

ETIOLOGY

Chronic laryngeal stenosis may be congenital, inflammatory, traumatic, neoplastic or neurogenic (Fig. 1). The latter two of these, neoplastic and neurogenic lesions, will be included in this discussion only when surgical or irradiation procedures have been followed by chronic laryngeal stenosis that has necessitated endoscopic or external surgical corrective procedures.

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Congenital laryngeal stenosis is found in the supraglottic, glottic and subglottic portions of the larynx. While in the past webs or bands stretching across the glottic chink, uniting the cords, were considered the commonest site of congenital stenosis,¹ study of our own series suggests that congenital subglottic stenosis is much more commonly encountered than are the webs² (Fig. 1A). Nineteen patients with congenital webs and one with a total glottic atresia were observed of whom four required tracheotomy. During the same period, 34 cases of congenital subglottic stenosis were seen of whom 14 required tracheotomy, and there were two cases of supraglottic stenosis, one of whom required a tracheotomy.³

Inflammatory lesions responsible for chronic laryngeal stenosis have fortunately decreased in frequency during the past twenty years.³ Of the acute inflammatory diseases of the larynx resulting in chronic laryngeal stenosis, diphtheria, laryngotracheobronchitis and scarlet fever were responsible for most of the destructive processes leading to laryngeal stenosis. Jesberg,⁴ in 1942, stated, "By far the most common cause of laryngeal stenosis is diphtheria." He reported the treatment and progress of 108 cases of postdiphtheritic laryngeal stenosis seen in a 22 year period. He also demonstrated statistically the rapidity with which diphtheria has decreased in incidence in the Los Angeles area during the period of his report, 1920 to 1942. Similar dramatic statistics are available for every metropolitan area, thus, fortunately, removing almost completely this serious etiologic factor of chronic laryngeal stenosis. Scarlet fever, too, has decreased in frequency and severity, with a lessened incidence of destructive laryngeal disease. At present the commonest acute laryngeal infection in infants and children is laryngotracheobronchitis. This condition was formerly responsible for many of the subsequent laryngeal stenoses since intubation was generally used to relieve laryngeal obstruction. With antibiotics and improved techniques of tracheotomy the incidence of postinfectious laryngeal stenosis from this disease is also remarkably lower than formerly.

Included in the etiologic category of inflammatory lesions are the postintubation and posttracheotomy stenoses that follow the surgical efforts employed to relieve an acute laryngeal obstruction. While these might be considered as traumatic, they are classified here since the basic etiology was the acute inflammatory disease. According to Jackson⁵ the high tracheotomy, which was generally an incor-

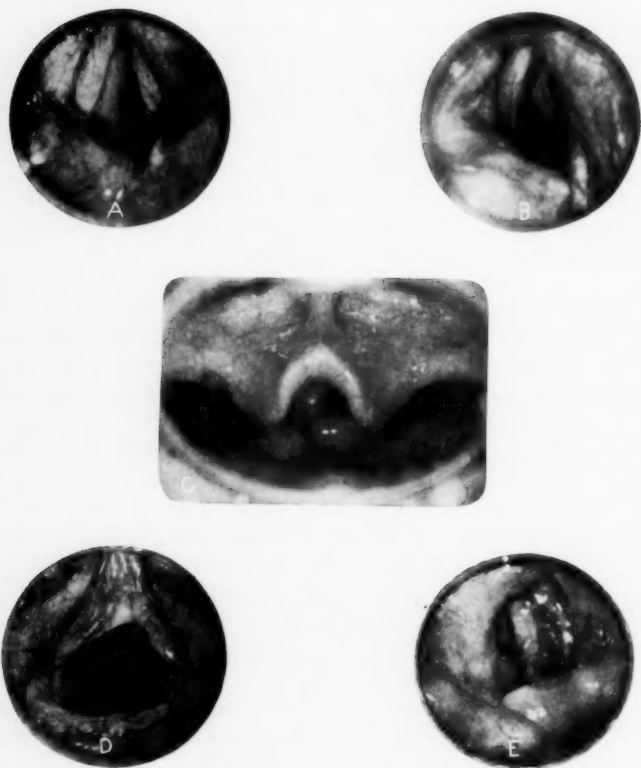


Fig. 1.—Mirror and direct laryngoscopic photographs of five cases of chronic laryngeal stenosis. A. Congenital subglottic stenosis in an infant four months of age. B. Deformity and stenosis of the larynx of a 21-year old woman intubated for several weeks in childhood for diphtheria. C. Compression fracture of the larynx incurred in an automobile accident. The arytenoids are dislocated and pushed forward, the epiglottis lies back and the airway is closed. D. Synechia causing partial stenosis following the removal of polypoid tissues from both cords simultaneously. E. Total occlusion of the larynx with fixation of the arytenoids following cautery of laryngeal papillomas.

rectly performed tracheotomy, was the principal cause of the subsequent stenosis. Robb⁶ showed that expert intubation and tracheotomy, the latter performed tranquilly after the airway was established by intubation, was followed by only one case of stenosis in 603 patients intubated, tracheotomized or both for laryngeal diphtheria.

In the past ten years, 30 patients with chronic laryngeal stenoses have been seen who had been intubated, tracheotomized, or both for acute laryngeal obstruction (Fig. 1B). Five were children, the remaining 25 were adults who had had their original infection in infancy or childhood, followed by the voice changes, respiratory obstruction and laryngeal finding of chronic stenosis. Most of the patients did not know the nature of the original infection, i.e., whether it was diphtheria or acute laryngotracheobronchitis. One followed scarlet fever, and one typhoid fever. Both had been intubated years previously. Four were due to acute abscesses of the larynx.

Specific chronic inflammatory lesions found to be responsible for chronic laryngeal stenosis are tuberculosis, syphilis, and rare cases of fungus disease. Rhinoscleroma,⁷ one of the commonest causes of laryngeal stenosis in some parts of the world, has fortunately been encountered in only three cases. There were 11 cases of luetic stenosis of the larynx, three of tuberculosis and in two cases pemphigus was responsible for the chronic laryngeal stenosis.

Trauma is rapidly replacing acute laryngeal disease as the commonest cause of chronic laryngeal stenosis. This includes internal laryngeal trauma as well as external trauma. Automobile accident injuries, cut throat and injuries from machinery and projecting pipes in industry represent external trauma. Postsurgical and postirradiation stenosis, and feeding tube stenosis comprise a second group that may be designated as intralaryngeal traumatic stenosis. Acute burns such as flash-burns of the respiratory tract and caustic burns represent a third type of trauma that results in chronic laryngeal stenosis.

Of particular importance are the problems associated with automobile accident injuries. The injury almost invariably occurs to the front seat passenger whose extended neck strikes the dashboard as the head is thrown forward through the windshield in a whiplash fashion (Fig. 1C); it may occur to a rear-seat passenger who is thrown forward against the back of the front seat. Some measure of



Fig. 2.—A. Lateral x-ray of the neck of the patient shown in Figure 1C. Note the obliteration of the airway and loss of cartilaginous landmarks. B. X-ray of patient shown in Figure 1E, laryngeal atresia following cautery of laryngeal papillomas.

protection is afforded by a safety belt,⁸ and a heavy sponge rubber pad over the dashboard; but while these suggestions are recommended by those attempting to reduce morbidity and mortality from auto accidents, few heed this advice.

In the present series, there are 14 cases of automobile accident fractures of the larynx and one identical fracture that was incurred in the landing of an airplane. Eight were males, seven females, ranging in age from 16 to 47 years. In ten the fracture was old and the cartilages were deformed and fixed. In only five was the accident recent, permitting prompt therapy which prevented excessive deformity.

Other external trauma responsible for chronic laryngeal stenosis in this series consisted of five gunshot wounds, three cut-throat in-

juries, and one each of golf-ball blow, baseball bat blow, a blow from a first incurred in boxing, striking a wire, striking machinery, and entanglement of a scarf in a tractor.

Internal trauma resulting in stenosis was largely iatrogenic. While there were four cases of chronic laryngeal stenosis resulting from caustics, there were eight due to the surgical removal of bilateral benign polyps (Fig. 1D), nine following the removal of papilloma. In five of the latter two groups of 17 patients, cautery had been used, or the procedure was followed by intra- or extralaryngeal irradiation (Fig. 1E). Certainly, the severity of the stenosis in these five is evidence enough to condemn the practice of indiscriminate intralaryngeal cautery, and radium or radon insertion or external radiation for benign lesions. The stenosis seen following single stage bilateral polypectomy in chronic bilateral hyperplastic polypoid laryngitis should also be sufficient warning that it is best to remove the polypoid tissue from only one side at a time. The stenosis that followed irradiation for papilloma was slowly progressive, increasing to the point at which relief was sought 15 to 30 years after the radiation had been administered.

Feeding tube stenosis of the larynx was first described by Iglaner and Molt.⁹ Since then many additional cases have been reported in the literature. A review of the succeeding cases reported with a description of our own experience¹⁰ added four cases to the 24 previously reported. This subject was again reviewed in 1958 by Strohl, et al.¹¹ Seven additional cases are found in reports by Robb,⁶ Figi,¹² and Hippenmeier,¹³ and thus it is obvious that this subject is more than a medical curiosity, and is in reality an important clinical problem.

Feeding tubes were found to be responsible for five of our cases of chronic laryngeal stenosis. The pathogenesis is apparently a trophic ulcer of the cricoid cartilage due to pressure of the tube lying between the cricoid plate and the cervical spine. Subsequent perichondritis, chondritis and ultimate destruction and collapse of the cricoid cartilage is responsible for the stenosis. The complication, which often requires wearing a tracheotomy tube for months or years, can be avoided by shifting the position of the feeding tube in the patient's throat or removing it entirely if it causes pain. Failure to recognize the correlation between the use of the feeding

tube and laryngeal stenosis may be due to the fact that usually the collapse of the larynx follows the use of the tube by six weeks to three months.

SYMPTOMS AND DIAGNOSIS

Progressive stridor, prolonged, noisy inspiration and expiration, dyspnea, supra- and infrasternal retractions and mental changes of chronic anoxemia are associated with the airway obstruction in chronic laryngeal stenosis. Voice changes range from variations in pitch and decrease in volume to total aphonia and the development of a buccal voice produced by articulating with the air forced between the cheeks and teeth for phonation.

Palpation often discloses deformity, dislocation, abnormal movement or fixation of the laryngeal or cricoid cartilages, or the supporting thyrohyoid membrane. Quite commonly in traumatic fractures of the larynx the thyroid cartilage is flattened or one ala is found to overlap the other anteriorly. In recent injuries, crepitation and abnormal motility are noted; in old injuries, abnormal fixation and induration suggest deformity sufficient to cause stenosis.

X-ray studies of the larynx, even in the infant, are of inestimable value when chronic laryngeal stenosis is suspected, not alone for diagnosis but for record and subsequent progress studies. Lateral x-rays of the neck for soft tissues often suffice (Fig. 2), but in questionable cases, or for more detailed study, the anteroposterior intra-esophageal film studies as well as planographic studies are indicated. The injection of opaque media such as Lipiodol® is occasionally helpful.

Mirror and direct laryngoscopy with photographic records permit the most accurate analysis of the problem of management of chronic laryngeal stenosis. These studies permit not only before and after comparisons (Fig. 3), but also a chronological record (Fig. 4).

Final evaluation of the general configuration, the location, degree and extent of a chronic laryngeal stenosis must be made by direct laryngoscopic and tracheoscopic examinations. For such purpose, the smooth-tip, C. L. Jackson laryngoscope and tracheoscope permits introduction of the instrument through the glottis and into the usually distorted subglottic area and finally into the trachea. In case

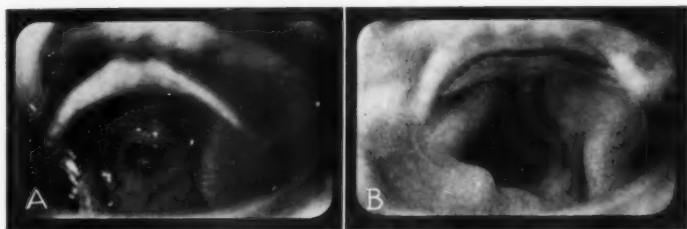


Fig. 3.—Mirror views of chronic laryngeal stenosis, (A) before and (B) after surgical reconstruction. The patient was the front seat passenger in an automobile collision.

of severe stenosis or total atresia, examination may be successfully made with the child-sized or even infant bronchoscope introduced through the mouth or from below upward through the tracheostomy.

TREATMENT

Obviously, the best treatment of chronic laryngeal stenosis is to avoid procedures that have been shown to cause it, and to employ procedures that will prevent its development following acute injury. Much has been done in this regard and more can be done. Certain fairly common causes of laryngeal stenosis have been effectively removed. For example, tuberculous and luetic stenoses are now seen only very rarely, and generally these are old, long-standing cases. Similarly, diphtheria, a frequent cause of stenosis in the past, is now rarely seen. Furthermore, intubation with the O'Dwyer tubes for either diphtheria or acute laryngotracheobronchitis has largely been supplanted by early, tranquil tracheotomy. Laryngeal abscesses have been reduced in frequency because of antibiotics, and when they do develop generally respond to antibiotic management both before and after a needed tracheotomy. Other direct prophylactic measures consist of low rather than high tracheotomies to avoid injury or infection of the cricoid cartilage; the substitution of a tracheotomy for prolonged, postoperative intratracheal intubation; and a gastrostomy to obviate the necessity of prolonged use of an in-dwelling nasogastric tube.¹¹

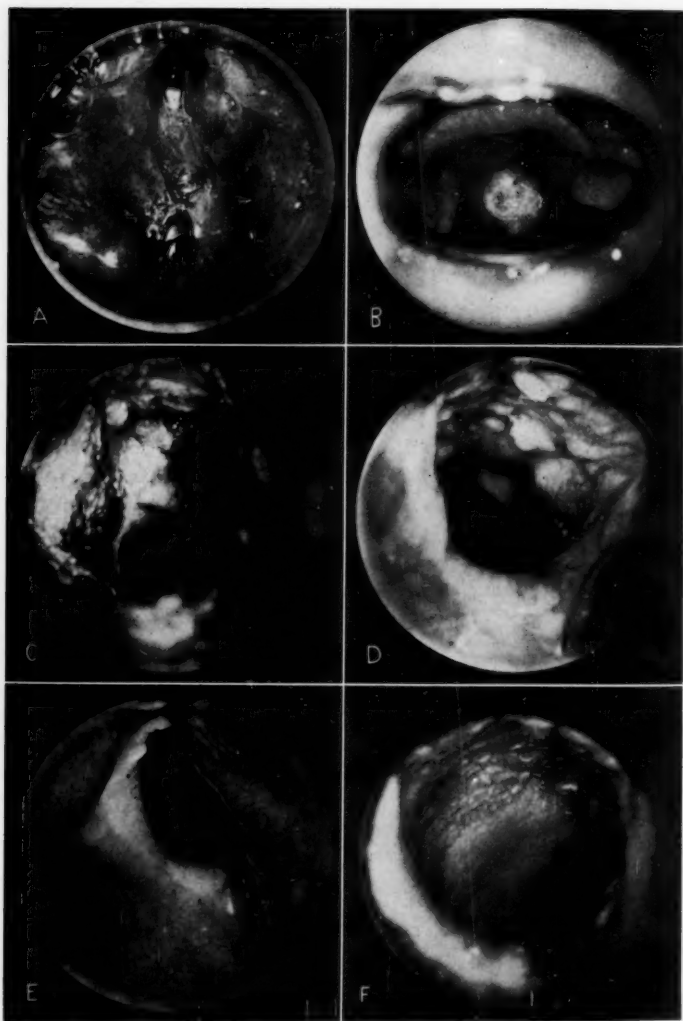


Fig. 4.—Laryngeal photographs showing successive stages during surgical reconstruction of the larynx. A. Direct view of the larynx shown in Figure 3A. Only a pinpoint opening remains. The contour of the cords is entirely lost and the normal structures are replaced by scar tissue. B. Mirror view of the larynx ten days after insertion of the Stent graft through a thyrotomy. The graft is seen within the larynx. C and D. The interior of the larynx immediately after endoscopic removal of the foam rubber Stent mold. E. Mucocutaneous junction at the entrance of the larynx nine months later after permanent removal of the polyethylene obturator. F. Skin lining the interior of the larynx.

Acute automobile-accident injuries to the larynx require as active a therapeutic regime as would be employed in an acute crushing injury of the nose. Broken cartilages must be replaced and the airway re-established and splinted, with support maintained until healing is effected.¹⁴ Emergency tracheotomy is life-saving when the accident has occurred and the compressed laryngeal structures occlude the airway. It is extremely important that the tracheotomy be placed low, as far from the fractured larynx as possible to avoid loss of the cricoid through further trauma and infection. The active measures that should be undertaken early should restore and maintain the fractured cartilages as close to their original position as possible as soon after the accident as is permitted by the general condition of the patient. Peroral manipulation with forceps or a laryngoscope tip may suffice in some cases to re-establish the airway. In other cases immediate open reduction may be required to obtain satisfactory realignment of cartilages. Following either procedure, a polyethylene or similar tube may be inserted into the glottis to serve as a splint to support the soft tissues until healing takes place (Fig. 5).

It is essential that these procedures be instituted within the first few days following the accident before the cartilages have become fixed in their compressed, obstructing position; and before scar tissue forms in the accompanying hematoma to further contract or even completely obliterate the airway. These procedures are too often delayed in the hope that as swelling subsides the airway will return to an adequate size. This cannot occur unless the trauma has been minimal and unaccompanied by fracture-dislocation of the cartilaginous framework of the larynx.

Fracture of the larynx in infants and children as well as the management of any chronic laryngeal stenosis in this age group present individual problems. Jackson and Jackson¹⁵ have stressed growth of the larynx as a factor in treatment. Increase in the size of the larynx during puberty can be used to advantage with the aid of increasing sizes of smooth rubber tubing which exert a constant, gentle internal pressure. They point out the fallacy of the statement, "He will outgrow it," referring to the laryngeal stenosis, urging that in cases of lesser severity, the tracheal canula be partially corked to force the child to use the larynx for respiration.

The management of the established late case of chronic laryngeal stenosis depends on the degree of respiratory impairment and the

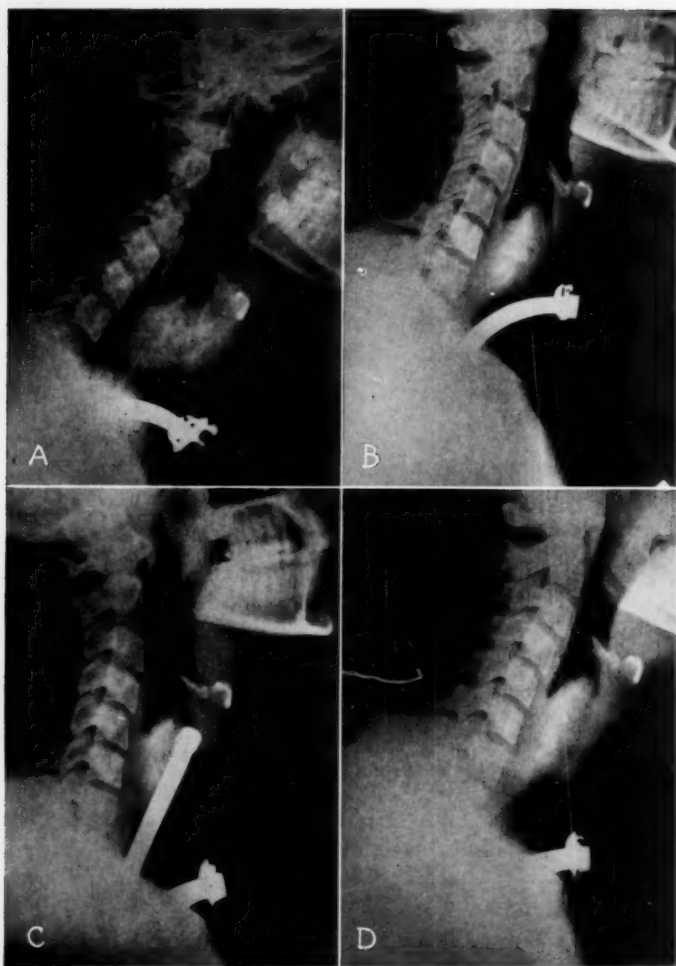


Fig. 5—A. Lateral x-ray of the neck of a 21-year old man shortly following an automobile accident. The crushed, swollen larynx, extensive cervical emphysema, obliteration of the airway and high tracheotomy are shown. B. X-ray demonstrating partially healed, stenotic larynx three weeks later. C. Tracheotomy tube lowered and intralaryngeal splint inserted to re-establish the airway and maintain the position of the cartilages. This step should have been taken as early as possible after the accident, but fortunately, patency of laryngeal airway was re-established. D. X-ray prior to removal of tracheotomy tube.

nature of the obstruction, as well as on the age of the patient. The treatment of many cases is effectively accomplished by simple endoscopic dilatations. This direct laryngoscopic procedure is done without anesthesia in children and under local anesthesia in adults. Round or triangular bougies with rounded edges are equally effective and often give excellent results even in patients who have had a moderate stenosis over a long period of time. The use of a Knight¹⁶ laryngeal bougie and knife blade is often of practical value in this procedure. Both respiration and speech are improved by dilatation and, if begun cautiously, the patient may not require a tracheotomy.

Other strictly endoscopic procedures consist of the peroral insertion of intralaryngeal dilators that are fixed in the larynx by various types of attachment to the tracheotomy tube. The classical work of Schmigelow¹⁷ was one of the early contributions to this problem. Rubber tubing and the rubber core molds suggested by Chevalier Jackson¹⁸ were used successfully for many years.¹⁹ An excellent review of the early literature of the subject is given by Clerf²⁰ in his discussion of cicatricial stenosis of the larynx. Rubber molds have been replaced to some degree by less irritating acrylic molds.²¹ Steinmann²² follows Erich's technique, using a dental molding compound to fit the acrylic mold to the individual patient. Cardwell²³ describes the use of an acrylic mold formed from a cadaver larynx, not attached to the tracheotomy tube, but it was suggested that such attachment would be advantageous. Salem²⁴ has prepared the acrylic mold in such a manner that the tracheotomy tube passes through an opening in its base.

The multitude of procedures suggested for management of chronic soft-tissue anterior commissure webs and stenoses indicates the difficulty of the problem. McNaught²⁵ suggested the use of a tantallum shield, fixed to the thyroid cartilage. This strip of metal placed between the split, cut edges of the cords keeps them separated long enough to permit epithelialization. Of course, this method can be used only if the cartilaginous laryngeal box is normal in size and the stenosis is primarily glottic.

Surgical reconstruction of the completely stenosed larynx becomes necessary when the lumen is obliterated and the configuration of the supporting cartilages has been destroyed by trauma or infection (Fig. 3). The surgical steps are begun only after evidence of



Fig. 6.—X-rays showing successive stages in the repair of an auto fracture of the larynx (see Figure 3). The patient had had no early therapy for the laryngeal fracture other than the tracheotomy. A. Lateral x-ray of the neck when the patient was first seen, two years after the accident. Complete laryngeal atresia is apparent. The direct laryngeal photograph taken at this time is seen in Figure 4A. B. Stent graft and temporary feeding tube. The wire fixation suture is seen (see Figure 4B). C. Foam rubber stent removed and replaced by a polyethylene obturator (Figure 8). D. Final x-ray prior to removal of tracheotomy tube. This x-ray was taken shortly before the direct and mirror views shown in Figures 4E, 4F and 3B.

perichondritis has disappeared, and all methods have been used to reduce or eliminate sources of infection.²⁶ Negus²⁷ refers to skin grafting and the use of a stent graft in patients in whom the cords adhere together over a wide extent, but in whom there is not much destruction of cartilages. The obturator is left in place a week to ten days. With destruction of cartilage the tendency to collapse necessitates the wearing of an obturator for some months. Figi¹⁴ and Erich²¹ further elaborated the technique, employing the stent graft. This practical, effective procedure is generally reserved for the most severe cases that cannot be given a normal airway by other more conservative means. Scar tissue and deformed obstructing cartilage necessitates removal of most of the tissue within the larynx. After the skin graft has been satisfactorily placed, months of careful post-operative care and the use of an acrylic or other obturators are necessary to prevent reformation of the stenosis. Figures 4 and 6 illustrate the successive steps in surgical reconstruction of the larynx shown in Figure 3.

Prior to the actual reconstruction, the previously performed tracheotomy may have to be revised to lower it sufficiently from the larynx to prevent it from affecting the operating field. High tracheotomy has always been considered one of the important etiologic factors of chronic laryngeal stenosis. Undoubtedly, this is true, but it is always implied that the tracheotomy had been performed "high," cutting through the first or second rings, the cricoid cartilage or through the cricothyroid membrane. Observations made on patients in this series of chronic laryngeal stenosis indicate that often the tracheotomy tube wanders upward if the tube is worn for several months. Replacing the tube to a "low" position in the suprasternal notch is no assurance that it will remain in that position. This is undoubtedly due to the position of the tapes holding the tube in place, particularly if the larynx lies low in the neck and the neck slopes excessively on the sides. In one patient, the tube had to be surgically replaced to a low position three times during the course of management of the stenosis. Finally, a second tape was passed downward under the arms and tied in the back in addition to the conventional tape through the tube and around the neck. The low position of the tube was thus maintained until treatment was concluded.

The first step in reconstruction consists of opening the larynx, establishing an adequate airway from the pharynx into the trachea

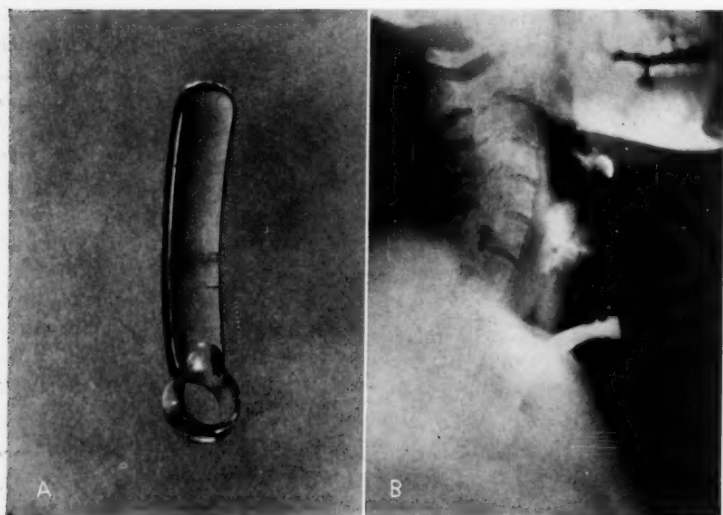


Fig. 7.—A. Acrylic laryngeal obturator with opening for tracheotomy tube. B. Lateral x-ray of the neck showing obturator in place. Tracheotomy tube passed through the opening holds it in place.

and preparing the tissue bed for a stent graft. A collar incision at the level of the lower border of the thyroid cartilage is preferred, although often the excision of a previously made high midline tracheotomy scar makes necessary a midline exposure from the hyoid bone to the tracheotomy opening. After exposure of the deformed thyroid cartilage, it is opened in the midline with a scalpel or turbinatome, although in old fractures sufficient deformity and ossification may be present to necessitate use of an electric saw. The pharyngeal and tracheal openings are then exposed and excess scar, deformed cartilage and often the dislocated, obstructing arytenoids are removed. Bleeding is controlled with fine ligatures and gelfoam to provide a smooth, dry bed for the stent graft. Since the compression fracture and the surgical step just described causes some loss of support for the epiglottis, there may be a tendency for this structure to fall posteriorly, partially occluding the airway after reconstruction has been completed. For this reason it is advisable to use one or more sutures at

this time to lift it anteriorly, fixing it securely to the base of the tongue.

The second step in the surgical reconstruction consists of preparation of the stent graft. A foam rubber mold is prepared which will fit into the laryngeal bed with a moderate degree of tension. A one-half thickness graft is then taken from a hairless donor site and sutured around the entire foam rubber mold, raw surface outward. Next the mold is placed in the larynx, the larynx closed with interrupted sutures and the mold held in position by an external wire suture passed through the skin, the thyroid cartilage and the mold. In the preparation of the stent graft we are indebted to Dr. Paul Greeley, Dr. G. Kenneth Lewis and Dr. Clarence Monroe.

The stent graft remains in place for ten days (Fig. 4B), following which the third step in the surgical reconstruction begins. The foam rubber mold is removed from the larynx by direct laryngoscopy. As the larynx is exposed with the laryngoscope, the rubber mold is grasped with forceps and withdrawn as the wire transfixation suture is cut and removed (Figs. 4C and D). Then, in order to maintain the skin-lined lumen thus constructed, acrylic or polyethylene molds are inserted (Figs. 7 and 8). The polyethylene tubes are held in place by braided silk threads fixed to the tracheotomy tube, or they may be prepared individually as suggested by Erich.²⁰ They are changed every six weeks and used for a period of six to nine months or until it is apparent that the lumen is adequately maintained (Figs. 4E and F) after a trial period of one to two weeks during which the obturator is left out (Fig. 6D). If the larynx shows no tendency to close during this trial period, the tracheotomy tube is removed and the tracheotomy closed (Fig. 3B).

Of the 13 patients with laryngeal atresia or chronic laryngeal stenosis thus treated, ten have had a satisfactory restoration of respiratory function through the larynx and closure of the tracheostomy. Two are still undergoing treatment with the polyethylene molds in place attached to their tracheotomy tubes. The thirteenth patient has a serviceable voice and an adequate airway but while she can eat solids and semi-solids without difficulty, aspirates liquids in attempting to drink. Control of this aspect of deglutition is improving.

All patients are referred to the Division of Speech for study, voice recording and instruction. Improvement in vocal function is

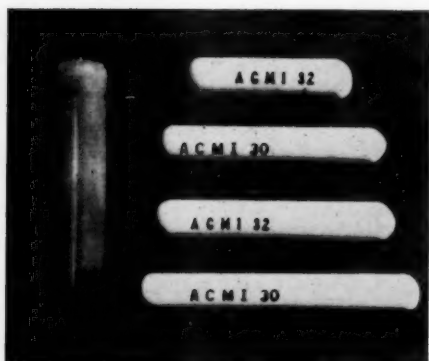


Fig. 8.—Laryngeal obturators of polyethylene tubing. Suitable size and length can be determined by x-ray measurements. The obturators are fixed to the tracheotomy tube by a heavy silk thread (see Figures 5C and 6C).

obtained through the determination of the most efficient utilization of pharyngeal and laryngeal muscles or scar tissue bands. Physical as well as psychologic benefits are very apparent in these patients through group lessons when they are in contact with others who have difficulty in expression similar to their own.

SUMMARY AND CONCLUSIONS

A series of 163 cases of chronic laryngeal stenosis treated during the past ten years is reviewed. Sixty-one were congenital in origin; 21 glottic, 3 supraglottic and 37 subglottic. Four cases were due to acute laryngeal abscesses. In 43 cases the etiology was a specific inflammatory process, 11 were luetic, 3 tuberculous, 3 were due to rhinoscleroma, and one was caused by typhoid fever. Two were due to pemphigus. In 21 cases of severe chronic stenosis the etiology was diphtheria, laryngotracheobronchitis or scarlet fever with acute laryngeal obstruction necessitating intubation or a tracheotomy or both. These patients were seen from six months to 24 years after the acute process.

Fifty-seven cases were traumatic in origin. Fourteen resulted from automobile accidents, one a tractor and one was incurred in an airplane accident. Five followed gunshot wounds of the neck, 3 resulted from cut-throat injuries, 4 the ingestion of caustics, 3 striking machinery, one patient struck a wire, one was struck by a baseball bat, one by a golf ball, and in one patient the injury incurred in boxing. Stenosis due to surgical trauma consisted of bilateral endoscopic procedures for benign lesions in 17 cases, in which cautery or postoperative irradiation was used in 5. Five followed the use of indwelling nasogastric tubes. Bilateral paralyses following thyroidec-tomies and the malignancies are not included in this series.

Voice therapy was used extensively in this series, improving phonation and respiratory control. Twenty-seven patients were seen for diagnosis only, did not require treatment or refused treatment. Tracheotomies were present or were performed in 138 cases during the course of treatment. Dilatations alone were used in 123 patients with or without trachotomy; this includes the 37 cases of subglottic stenosis and other stenoses in infants who received occasional dilatations during the normal growth of the larynx from its infant to child size. Thirteen patients required external surgical reconstruction with the use of a split-thickness stent graft.

The management of chronic laryngeal stenosis must be directed toward re-establishment of the normal airway, restoration of a serviceable voice and insuring a functional result that permits satisfactory closure of the larynx during deglutition.

Chronic laryngeal stenosis due to specific and acute inflammatory diseases is decreasing in frequency. Congenital lesions, and external and internal trauma have replaced the inflammatory processes in etiologic significance. External trauma is incurred most frequently in automobile and industrial accidents. Internal trauma resulting in laryngeal stenosis may follow the use of indwelling feeding tubes and anesthetic intratracheal tubes, and bilateral endoscopic surgical procedures, particularly if cautery or postoperative irradiation has been used.

Symptoms are phonatory and respiratory. The former vary from dysphonia to total aphonia. The latter range from mild stridor to complete laryngeal atresia. The diagnosis is established by the

palpation of cartilaginous deformity, x-ray evidence of airway narrowing and cartilaginous destruction, and mirror and direct examinations of the larynx. These demonstrate the cicatricial changes and limitation of motion characteristic of chronic stenosis. Mirror and direct laryngoscopic photographs of the larynx provide important records of progress of therapy and a careful analysis of the effectiveness of treatment.

Minimal chronic laryngeal stenosis requires only occasional dilatation of the larynx to maintain an adequate airway. In more severe stenosis, repeated peroral dilatation may develop a satisfactory airway. The peroral insertion of fixed intralaryngeal dilators or core molds of rubber or acrylic is also effective. External laryngeal procedures are used in more severe stenoses. In one procedure, metal implants are fixed to the thyroid cartilage and inserted between the cords to permit epithelialization of the cord edges. For more severe, destructive processes, exenation of the intralaryngeal scar tissue and the insertion of a stent split-thickness skin graft is necessary. With severe loss of cartilage, the stent graft may require additional support obtained with costal cartilage.

The most effective treatment of chronic laryngeal stenosis is to prevent its occurrence; tracheotomy rather than intubation; low rather than high tracheotomy; removal of feeding tubes if they cause a sore throat; early and adequate therapy of acute laryngeal abscesses, tuberculosis and syphilis; and in crushing injuries (automobile accidents), replacement of laryngeal cartilages and support with intralaryngeal splints until healing takes place.

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REFERENCES

1. Clerf, Louis H.: Congenital Stenosis of the Larynx: Report of Three Cases. *ANNALS OF OTOLGY, RHINOLOGY AND LARYNGOLOGY* 40:3:770 (Sept.) 1931.
2. Holinger, Paul H., Johnston, Kenneth C., and Schiller, Filmore: Congenital Anomalies of the Larynx. *ANNALS OF OTOLGY, RHINOLOGY AND LARYNGOLOGY* 63:3:581 (Sept.) 1954.
3. Bigler, John A., Holinger, Paul H., Johnston, Kenneth C., Schiller, Filmore: Tracheotomy in Infancy. *Pediatrics* 13:5 (May) 1954.
4. Jesberg, Simon: Postdiphtheritic Laryngeal Stenosis. *J.A.M.A.* 1000-1002 (Nov. 28) 1942.
5. Jackson, Chevalier: High Tracheotomy and Other Errors the Chief Causes of Chronic Laryngeal Stenosis. *Surg., Gynecol. and Obstet.* 392-398 (May) 1921.

6. Robb, James Milton: Laryngeal Stenosis. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 58:2:566 (June) 1949.
7. Holinger, Paul H., McMahon, Robert J., Johnston, Kenneth C.: Rhinoscleroma. *Ill. Med. Jour.* 103:6 (June) 1953.
8. Woodward, Fletcher D.: General Medical Aspects of Automobile Crash Injuries, Deaths. *J.A.M.A.* 163:4:225-259 (Jan. 26) 1957.
9. Iglauer, Samuel, and Molt, William F.: Severe Injury to the Larynx Resulting from the Indwelling Tube: Case Reports. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 48:886, 1939.
10. Holinger, Paul H., Loeb, William J.: Feeding Tube Stenosis of the Larynx. *Surgery, Gynecol. and Obstet.* 83:253-258 (Aug.) 1946.
11. Strohl, E. L., Holinger, Paul H., Diffenbaugh, Willis G.: Nasogastric Intubations: Indications, Complications, Safeguards and Alternate Procedures. (In Press)
12. Figi, Frederick A.: The Etiology and Treatment of Cicatricial Stenosis of the Larynx and Trachea. *South. Med. Jour.* 40:1:17-26 (Jan.) 1947.
13. Hippenmeier, Von W.: Perichondritis Laryngea nach Verwendung von Verweilsonden in Oesophagus bei Magenulcera. *Schweiz. Med. Woch* 76:170, 1946.
14. Figi, Frederick A.: Chronic Stenosis of the Larynx with Special Consideration of Skin Grafting. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 49:2:394 (June) 1940.
15. Jackson, Chevalier, Jackson, Chevalier L.: Laryngeal Stenosis, Growth of the Larynx as a Factor in Treatment. *The Laryngoscope* (Nov.) 1932.
16. Knight, J. S.: Laryngeal Dilating Knife. *Tr. Am. Acad. of O. and O.* 59: 406 (May-June) 1955.
17. Schmiegelow, E.: Stenosis of the Larynx: A New Method of Surgical Treatment. *Arch. Otolaryngol.* 9:347 (May) 1929.
18. Jackson, Chevalier: Stenosis of the Larynx. *Trans. of Amer. Laryngol., Rhinol. and Otol. Soc., Inc.*, 1936.
19. Patterson, Ellen J.: Laryngeal Stenosis in Children with Special Reference to Treatment with Core Molds. *Arch. of Otolaryngol.* 29:71-77 (Jan.) 1939.
21. Clerf, Louis H.: Cicatricial Stenosis of the Larynx. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 42:1:1 (Mar.) 1953.
22. Steinmann, Von Erik P.: Zur Verwendung von Knuthharzen bei der Behandlung von Trachealstenosen. *Praxis*, No. 11 (Mar.) 1949.
23. Cardwell, Lt. Col. Edgar P.: Stenosis of the Larynx of Limited Extent: Simplified Treatment. *Arch. of Otolaryngol.* 44:560-564 (Nov.) 1946.
24. Salem, Waldemir: Estenose Cicatricial Extensa Da Laringe, Separata de "O Hospital", Rio De Janeiro, 1950.
25. McNaught, R. C.: Surgical Correction of Anterior Web in the Larynx. *Laryngoscope* 60:264-272 (Mar.) 1950.
26. Le Jeune, Francis, and Owens, Neal: Chronic Laryngeal Stenosis. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 44:354-363 (June) 1935.
27. Negus, V. E.: Treatment of Chronic Stenosis of the Larynx with Special Reference to Skin Grafting. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 47:4:891 (Dec.) 1938.
28. Looper, Edward A.: Use of the Hyoid Bone as a Graft in Laryngeal Stenosis. *Arch. of Otolaryngol.* 28:106-111 (July) 1938.

XXXIX

EVIDENCE OF LARYNGEAL PARTICIPATION IN EMOTIONAL EXPRESSION: ITS RELATION TO HYSTERICAL APHONIA

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During the Franco-Prussian War, Frisch and Hitzig demonstrated that stimulation of the surface brain cortex in man (with open skull injuries) produced movements of the contralateral extremities. From this time on the existence of a motor cortex has been recognized. The region (area 4) in front of the central fissure, generally recognized as the primary motor area, has often been stimulated with typical responses during brain operations for various intracranial lesions. The existence of a corresponding motor field has been demonstrated repeatedly by those working with experimental animals. The pattern for man and in a somewhat more simplified form for many mammals is generally accepted.

Stimulation of the paracentral gyrus on the medial surface of the hemisphere produces movements of the contralateral lower extremity beginning with the toes and then through ankle, legs, and thigh. Movement of the contralateral thigh or hip, for there is some individual variation, results from stimulation at about the point where the medial surface goes on over to the lateral surface of the hemisphere. Following down along the lateral surface of the hemisphere, the hip area is followed by a very narrow trunk area and this in turn by regions which successively give contralateral shoulder, arm, elbow, forearm, wrist, hand, finger (often individual finger) and thumb movements. Below the region having to do with thumb movements, and in the lower third of the motor area, is the area which gives movements of various parts of the face when stimulated. In the monkey, this is an area that gives movements of the homolateral face (Lauer 1952) and, at a point in this area, voluntary movements of

the larynx can be demonstrated (Furstenberg 1956). Conjugate deviation of the eyes may be obtained by stimulation at appropriate arm and face areas but the motor center for such movements is farther forward at the caudal end of the medial frontal gyrus. This pattern of motor responses is continued behind the central fissure into the sensory area with very little difference in specificity.

The discharge path from the motor area (and efferent portion of the sensory area) is the well known pyramidal system with its corticospinal and corticobulbar components. For the larynx, the corticobulbar fibers swing down with the rest of the pyramidal system into the internal capsule. Those related to laryngeal movements are in the posterior part of the genu of this capsule. From the internal capsule the fibers enter the middle three-fifths of the cerebral peduncle at the base of the brain. The corticobulbar fibers, including those which carry impulses to nucleus ambiguus for innervation of the muscles of the larynx, lie dorsomedial to the portion of the pyramidal system related to contralateral upper and then contralateral lower extremity movements. When the pons is reached the pyramidal fibers are broken up by crossing the fibers of the pontocerebellar system into smaller bundles, but the corticobulbar fibers, including those supplying the larynx, retain their relatively dorsal position. As the level of the trigeminal nerve is approached in the pons, the corticobulbar fibers related to laryngeal movements in part continue directly caudward and in part enter the medial lemniscus as aberrant fibers. Again, at facial nerve levels aberrant fibers are given off to become constituents of the medial lemniscus. Therefore, as the corticobulbar fibers concerned with facial movements reach the medulla, they are partly with the other pyramidal fibers in the pyramid and partly as aberrant fibers in the medial lemniscus. Within the medulla both the aberrant fibers and the corticobulbar component of the pyramidal system distribute bilaterally to the caudal end of the nucleus ambiguus in relation to the motor neurons which constitute the recurrent laryngeal and superior laryngeal nerves.

It is obvious, therefore, that ipsilateral lesions of either the cerebral cortex or of the supranuclear corticobulbar system projecting to ambiguus nuclei will not produce a paralysis of the larynx. Bilateral lesions of these cortical areas or their corticobulbar projection systems lead to a complete loss of voluntary laryngeal movements. So far as it is known, there is no difference in function between the

aberrant and the main corticobulbar fibers. Both systems are concerned with providing the pathways for voluntary motor control of the larynx. Since the distribution to the nucleus ambiguus is homolateral and contralateral, it is obvious that a supranuclear lesion which produces a paralysis of the larynx must be bilateral.

It is to be emphasized, however, that in addition to motor area 4 and the adjoining sensory areas related to the finer and more discrete voluntary movements of the larynx, information has been accumulating in regard to a number of other cortical areas which will produce contralateral, bilateral, or homolateral movements of parts of the body or the face when stimulated. These areas are termed second or additional motor areas. They are quite apart from the motor cortex and it can be demonstrated that their stimulation will elicit body or face movements even when the motor cortex bilaterally has been destroyed or the corticobulbar or corticospinal components have been severed.

However, the types of movements obtainable on stimulation of the second motor areas are relatively gross in character. Thus, for the upper extremity they are movements of a whole extremity, or of the shoulder and upper arm, or of the forearm and wrist, or perhaps the whole hand, but only rarely, if ever, of a single finger or a thumb. Moreover, they may be bilateral or contralateral (or from some areas homolateral).

On the face, stimulation of the second motor areas is represented by a closing of the eyes, or a generalized response which involves the face on the side of the stimulation, or on the opposite side depending upon the exact point of excitation. Deviation of the eyes and changes in size of the pupils may also be elicited from some second motor areas.

It is interesting to note that there is a pattern of movement on the second motor areas which follows a rather stereotyped plan. For example, if from a given portion of an area, stimulation produces contralateral or bilateral lower extremity movements, one obtains successively movements of the upper extremities, then of the face on the side opposite stimulation, and finally homolateral face responses in moving along the area. Movements of the eyes are usually associated with the portion of the area giving rise to homolateral face

movements or else independently located. The sequence may be from dorsal to ventral, or ventral to dorsal along the cortex or, as in the island region, from caudal to rostral.

It is to be noted that movements from the second motor areas are still elicitable (in some cases somewhat diminished) when the pyramidal tract is sectioned bilaterally. This suggests that the second motor areas have discharge paths other than the pyramidal system, although some of them may also discharge by way of the pyramidal tract.

The discharge paths from the second motor areas are multisynaptic paths. Some of them pass directly from the cerebral cortex to the tegmentum of the midbrain. An example of such a path is the corticorubral path from area 6 to the red nucleus. Others pass directly or by way of the basal ganglia to the tegmentum areas of the midbrain around the red nucleus where many of them synapse. From the red nucleus and the surrounding tegmental gray rubrobulbar, rubrospinal, tegmentobulbar, and tegmentospinal tracts carry the impulses back to the motor neurons of the cranial and spinal nerves. Many of these paths discharge bilaterally. These second motor paths provide the outlets for supplementary movements which augment the highly specialized voluntary responses and are in the nature of related cortical associated movements.

METHODS

In all these experiments the monkeys (*macaca mulatta*) were kept lightly anesthetized with ether. Additional motor responses cannot be obtained readily with barbiturates unless the experimenter is prepared to wait until the animal is under very light anesthesia. All the operations were performed with sterile technique and in each case the larynx was examined pre-operatively, during the operation, and postoperatively. The skulls were trephined with a dental drill to expose portions of the cerebral cortex which were to be stimulated. The dura was cut to expose the surface of the cortex. One electrode was used for stimulating while the indifferent electrode consisted of a rectal plug. The strength of the stimulation varied between five and twelve volts. The timing was 1 msec. The vibratory rate was 40 cycles per sec. Changes in respiration and motor responses accompanying the laryngeal movements were noted.

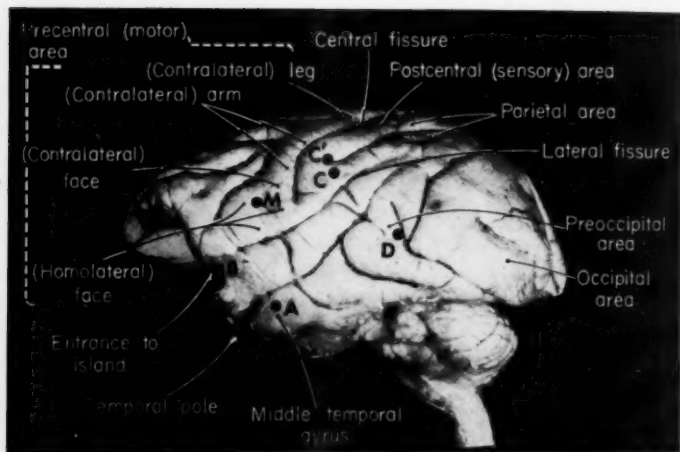


Fig. 1.—1. The center for voluntary movements of the larynx (Fig. 1M) in the monkey is situated in the precentral area which also gives voluntary movements of the homolateral face.

2. (a) Stimulation of point A in the middle temporal gyrus produced a closure of the larynx and also yielded homolateral movements of the face. With sudden closure of the glottic chink there were accompanying respiratory changes. (b) Similar responses were obtained from point B in front of the precentral gyrus on the under border of the frontal operculum. Also from C and C¹ in the parietal cortex and from D in the preoccipital cortex. (In some work done by Dr. Mary Jane Showers on stimulation of the cingulate area in the region rostroventral to the corpus callosum, the possibility of laryngeal movements and associated respiratory changes was documented.)

In all cases the laryngeal responses were obtained from portions of the second motor fields which gave movements of the lower part of the homolateral half of the face. These face movements were usually of a sneering type but chewing and tongue movements often were elicited near the area which showed laryngeal responses.

The movements of the vocal cords were observed by using a child's laryngoscope which had been modified for this purpose and laryngeal responses were photographed with the moving picture camera.

When the point was found which gave laryngeal responses when stimulated electrically and satisfactory pictures were obtained, this area was destroyed by fulguration. The destructive lesion was made somewhat larger than the point from which the laryngeal responses had been elicited but always with the center of the response area in the middle of the lesion. The increased size of the lesion as compared with the point of stimulation enabled us to identify the area in postmortem material with precision. Postoperatively, the animal was checked for abnormalities and allowed to survive on the average of three weeks in order that Marchi material could be prepared from the brain in case it was desired.

The animals were sacrificed by an overdose of ether or evipal and perfused with 10 per cent formalin. The brains were removed and placed in 10 per cent neutral formalin to obtain hardening before they were sectioned. The various points of stimulation and destruction have been plotted on the photograph of a monkey's brain (Fig. 1).

EXPERIMENTAL FINDINGS

Monkey F-C No. 1. The monkey was anesthetized (November 22, 1957) and the cortex of the middle and inferior temporal gyri exposed down to the temporal pole. The exposed area was stimulated. Through accident, a direct current damaged part of this area. However, when this was remedied, stimulation of the middle and inferior temporal regions on the lateral surface toward the rostral end of the temporal lobe produced homolateral face movements comparable to those demonstrated from this region by Schneider and Crosby (1954). These movements consisted of retraction of the upper lip on the left, closure of both eyes, and of the mouth. With the laryngoscope in position, stimulation of the temporal gyri disclosed a definite area which, when stimulated, gave movements of the vocal cords. The position of this area is indicated in Figure 1 at A. Because of the brain damage at the beginning of the experiment, it was necessary to use an unusually high voltage (12 volts; 40 cycles per second, 1 second delay, 1 millisecond duration). Photographs were taken and a lesion placed. The dura was closed in the usual fashion, the temporal muscle repaired and the skin margins approximated.

The animal reacted from the operation about 15 minutes after the anesthetic was stopped. For several days there was evidence of

edema but the animal was alert and active. Piloerection appeared the day after the operation and became marked when the animal was excited. During the succeeding days (up to December 12, 1957) the monkey gradually recovered from the operation although the piloerection remained.

On December 12, 1957, another operation was undertaken on the opposite side of the brain. The lateral fissure and the adjoining portions of the temporal and frontal lobes were exposed. The temporal and frontal opercula were gently retracted to reveal the front end of the island. Stimulation of the rostral part of the island (Fig. 1B) gave homolateral face movements. Stimulation at about the level of the rostral limit of the frontal operculum (point B) gave laryngeal responses and respiratory effects. Similar laryngeal movements and respiratory changes have been reported by Sugar, Chusid, and French (1948) who obtained movements of the vocal cords along with respiratory slowing or arrest, from the operculum rostral to the inferior portion of the precentral gyrus.

The animal recovered from the second operation in an uneventful manner. However, piloerection was still present. On January 14, 1958, a third operation was attempted. The brain was removed from the parietal area but the animal showed evidence of respiratory embarrassment and died from respiratory failure. Cardiac activity remained for approximately forty-five minutes under the influence of artificial respiration.

Monkey F-C No. 2. In order to confirm the results obtained on monkey F-C No. 1, a second animal, F-C No. 2, was operated upon on February 2, 1958, with exposure of the temporal pole and frontal areas. The frontal and temporal opercula were retracted carefully and homolateral facial movements observed from the lower half of the front end of the island (Fig. 1B). The respirations were rapid (50), the pulse strong and regular. On stimulation of the area rostral to the precentral gyrus where the island was opening out, there was apnea, gasping and forced respiration along with homolateral face movements including those of muscles of mastication. Through the laryngoscope it was noted that the vocal cords closed with apnea. When the lesion was placed, a compensatory decrease in respiration occurred.

When the temporal pole (Fig. 1A) was stimulated changes in respiration were again noted from the exact point where the laryngeal movements were obtained. As the circumference of the temporal pole was stimulated with the electrode, the animal made a noise like a growl. During the postoperative recovery period the respirations were shallow and jerky and the animal cyanotic and cold. The heart rate was rapid. When the animal had recovered sufficiently to be returned to his cage, it was repeatedly noticed that slightest exertion resulted in cyanosis about the face. Piloerection was marked. For several days postoperatively, there was indication of cyanosis but a return to a relatively normal state occurred in approximately two weeks. The animal was sacrificed and the brain perfused on March 3, 1958.

Monkey F-C No. 3. The monkey was operated upon on February 5, 1958, and the right temporal-parietal region exposed. Homolateral face movements were elicited from the cortex around the base of the interparietal fissure which is the transition region between parietal areas 5 and 7. These movements consisted of homolateral responses of the lip, tongue and jaw and bilateral closure of the eyes. On stimulation the respiration slowed to a gasping, shallow type and the glottis tended to close.

The preoccipital region was then stimulated in the area (Fig. 1D) from which homolateral face movements had been elicited previously (Lenmen 1954). The eyes turned down and toward the opposite side together with homolateral movements of the upper lip and the nasal area. Both pupils dilated, the larger one noted on the side contralateral to the stimulation. Stimulation in the region indicated at D on the figure resulted in closure of the glottis. Respiratory changes of an interrupted or gasping character accompanied the laryngeal response.

On January 7, 1958, the animal was reoperated with exposure of the parietal area on the left. The region around the base of the interparietal fissure at the transition between parietal areas 5 and 7 was stimulated as before. A closure of the eyes, an elevation of the ala of the nose, a movement of the upper lip, and a tensing of the jaw muscles resulted. As the stimulation was carried slightly farther dorsally, upper extremity movements were obtained. At the point indicated at C and C¹ in Figure 1 in this region which produced

movements of jaw and lip, stimulation brought about a closure of the vocal cords.

The animal's postoperative course was uneventful and he later was used for other experiments.

Monkey F-C No. 4. This monkey was operated upon on December 3, 1957 and the superior and middle temporal gyri exposed. The area was checked for homolateral face movements. The point at which movements of the larynx, along with face movements, could be obtained on stimulation was identified and marked by a lesion. Apparently the point at which best laryngeal movements were noted also gave particularly vigorous jaw and tongue movements. This animal was sacrificed December 30, 1957, after an uneventful postoperative period.

Monkeys M-C No. 1 and No. 2. The temporal region in these monkeys was explored for the possible existence of an area which would give laryngeal responses. The region stimulated was at A in Figure 1 for monkey M-C No. 1. The vocal cords closed but no respiratory changes were noted. Moving pictures were not taken.

Monkey M-C No. 2 had the temporal area exposed but an effort to verify the results obtained in monkey M-C No. 1 failed.

Monkey S-C No. 3. This monkey had a cingulate gyrus exposed and tested for additional motor responses. Laryngoscopic examination showed that movements of the larynx and changes in respiratory rate occurred in the region rostral and slightly rostroventral to the corpus callosum. This portion of the cingulate gyrus gives sneering movements of the face on the side of stimulation.

SUMMARY

From the preceding studies it is logical to make the following assumptions:

1. Closure of the vocal cords may be obtained from various regions of the brain other than the primary motor centers (Furstenberg 1937).

2. Some of the regions have directly or indirectly a stimulative action or an inhibitory effect on the areas of the hypothalamus concerned with emotional expression. One would expect then that an emotional drive might aid in producing laryngeal responses especially when primary supranuclear centers or paths are not functioning. Changes in respiration are parts of an emotional response.

3. Laryngeal movements may also be obtained from areas of the cortex (such as parietal and preoccipital regions) which do not appear to be related primarily to emotional responses. In such instances, they might be obtained in the absence of an emotional drive.

4. Laryngeal movements reported here are parts of additional motor patterns and occur only in regions where the stimulation also gives homolateral responses of the face and more particularly the tongue and lower jaw. It is to be emphasized that such laryngeal movements are noted from only very small areas of the region from which such homolateral face movements are elicited.

5. It is probable that laryngeal responses, like other gross body and extremity movements, are obtainable from additional motor areas in the absence of functioning primary motor cortices related to the larynx. Such additional motor areas for laryngeal responses provide a reserve mechanism for patients with bilateral destruction of the pyramidal system from the motor cortex as in a bilateral Weber's syndrome or in large pontine lesions. This must be true because the second motor paths course through levels of the brain for the most part independently of the pyramidal system. They cannot be destroyed therefore by common lesions.

6. The second motor areas probably function for automatic responses in association with changes of respiration and during the excitement or repression of emotional state. These second motor areas serve also to promote cortical automatic associated movements accompanying voluntary responses. (An example is whistling or humming while at work.) It should be possible therefore to use them as substitutes when supranuclear lesions cause laryngeal paralysis.

CONCLUSIONS

The foregoing results indicate that laryngeal movements can be obtained from many areas of the cortex in addition to the primary

motor field. These secondary motor areas have distinct and separate paths of multisynaptic character to lower motor centers (to the nucleus ambiguus for the larynx). It is obvious that such additional motor areas may be spared when the voluntary motor field or the pyramidal systems are destroyed. They represent then supplementary sources for possible laryngeal movements in cases of supranuclear laryngeal paralysis.

These secondary regions for laryngeal responses, so far as they have been demonstrated, were all located in the portions of the second motor fields which, on stimulation, gave homolateral face movements—particularly movements of lip, jaw, and tongue. Theoretically, therefore, it should be possible to train the larynx to function in patients with supranuclear lesions which have produced bilateral laryngeal paralysis. One might start with movements of the tongue in exploring this possibility. It would be tireless and stubborn training no doubt in view of the fact that the secondary motor fields would have to overcome the tonicity of cerebellar influence upon the vocal cords. In the monkey a lesion properly placed in the cerebellum should accomplish this effect.

Some of the additional motor areas are in fields where the connections suggest that they have to do with emotional responses. Consequently, they may function in emotional speech. Nielson tells of a British soldier who suffered a serious wound in the head and was rendered speechless. He was hospitalized in London. One day he saw a German airplane over the city about to drop bombs in a crowded district. As the soldier anxiously watched the bomber, anti-aircraft guns sighted it and shot it down. The soldier was heard to exclaim "Hallelujah" although he had not been able to speak at all since his injury and continued to be aphonic after this episode.

We have all seen patients with so-called hysterical aphonia who have been unable for weeks or months to speak above a whisper. In these cases there is obviously a central inhibitory effect upon the corticobulbar fibers carrying motor impulses to the nucleus ambiguus. When emotionally aroused, they may scream loudly and in a fit of passion have been known to speak audibly and distinctly. I have been consulted several times recently by a patient with hysterical aphonia which has been present for approximately one year. She speaks in a faltering whisper when attempting to carry on ordinary

converation but pronounces her words clearly and unhesitatingly if made to laugh during conversation. There are good reasons to believe that the lower motor neurons (nucleus ambiguus) are supplied by motor impulses from some of the second motor areas of the cortex in this group of patients.

It seems probable also that in the course of normal behavior the additional motor areas of the larynx, like those of other types of movements, serve as centers for automatic associated movements. They dress up and give personality as it were to the voluntary motor responses. Whistling, humming or talking to yourself (of which you are quite unaware) as you work might be an illustration of an automatic associated movement.

While second motor areas herein described have been definitely demonstrated in the cortex of monkeys, it is logically assumed phylogenetically that a similar pattern of motor innervation for the larynx is present and more highly developed in man.

UNIVERSITY HOSPITAL

A PANEL DISCUSSION

XL

REHABILITATION OF THE POST-LARYNGECTOMIZED PATIENT

I. TYPES OF CLINICAL CASES AND THEIR RESULTANT ESOPHAGEAL, PHARYNGEAL, AND NECK DEFORMITIES

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Deformities resulting from total laryngectomy and from operations which include laryngectomy depend in large part on the extent of the surgical removal. Thus one might consider separately the deformities following 1) laryngectomy without neck dissection, as practiced in intrinsic lesions unsuitable for partial laryngectomy but without palpable metastases, 2) laryngectomy with simultaneous neck dissection in continuity, as employed in intrinsic lesions with palpable nodes and in most extrinsic lesions with or without palpable nodes, 3) laryngectomy, partial hypopharyngectomy and neck dissection for lesions with unilateral involvement of hypopharynx and extrinsic larynx (Fig. 2a), and 4) laryngectomy, total hypopharyngectomy and neck dissection.

Common to all cases in which laryngectomy is a part of the procedure is the possible occurrence of 1) persisting pharyngeal fistula, 2) stenosis of the tracheal stoma, 3) stenosis of the upper trachea or 4) keloid.

Pharyngeal fistula is uncommon, though perhaps somewhat more often seen following T-closure of the pharynx than after linear closure, as well as in operations following previous irradiation. Careful approximation of mucosal edges with the first line of closure,

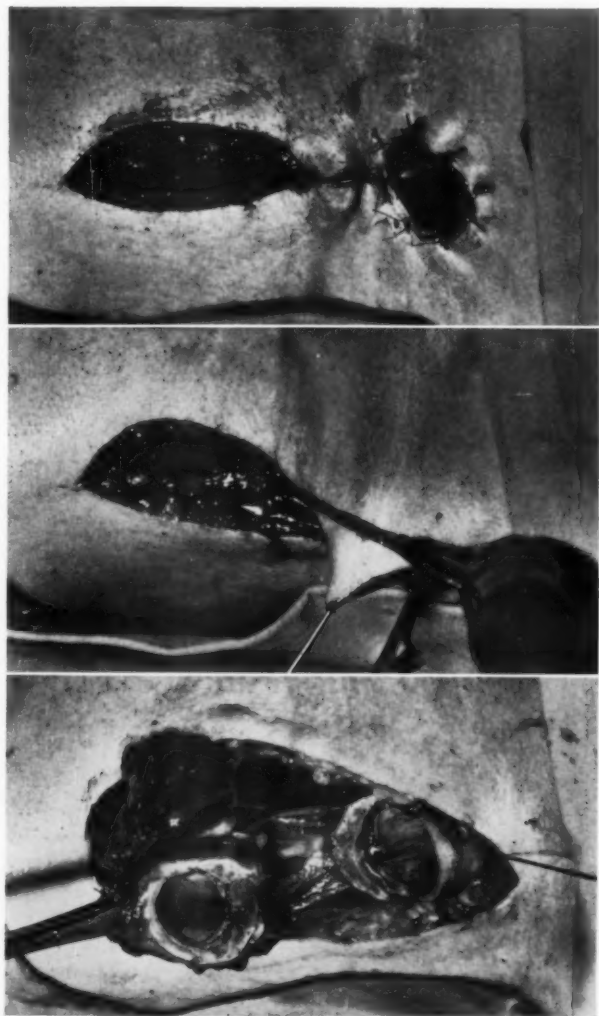


Fig. 1.—Construction of tracheal stoma in laryngectomy: a) completed stoma, b) technique of excision of skin at stoma for intrinsic carcinoma: c) conservation of segment of cricoid lamina.

inversion of the initial line with fine sutures including both constrictor muscle and mucosa and reinforcement by approximating the remaining portions of ribbon muscle and hyoid bone in such a way that all "dead-space" is obliterated appear to be the important items in technique favoring primary closure. Spontaneous closure is the rule if a fistula should occur, although occasionally secondary closure may be required.

Placement of the tracheal stoma well down in the suprasternal notch is an advantage, so that the opening will lie below the collar line in the depth of the jugulum.

Stenosis of the tracheal stoma is most easily avoided by preserving an annular lower section of the cricoid, or a segment of the lower portion of the cricoid lamina (Fig. 1a) if this is feasible. Excision of a moderate-sized hemi-ellipse of skin on either side of the lower portion of the skin incision (Fig. 1b) prior to approximation of skin-edges and trachea, oblique sectioning of the trachea, and transverse section of ribbon muscles at the level of the stoma may be helpful in preventing contracture. The use of a minimum number of stainless steel sutures (32 gauge) about the stoma appears to lessen the chance of infection in this part of the wound, which predisposes to secondary narrowing. A large cannula is usually worn during the immediate postoperative period (No. 7 to No. 9).

If a tendency to narrowing of the tracheal stoma should become evident, a regime of alternating intervals with and without the cannula may allow eventual decannulation. Otherwise, stenosis of the stoma may be corrected by revision, incorporating Z-plasty at the lower margin of the stoma, or by use of a hollow acrylic button (Fig. 2b) as described by Moore.⁸ The latter requires individual fitting and trial.

Stenosis of the trachea, below the level of the stoma, is usually the result of a poorly fitting cannula with persisting ulceration and cicatrization at the end of the tube. The tendency is increased if there is a decrease in the transverse diameter of the trachea due to poor support by tracheal rings, so that marked expiratory and tussive collapse occurs about the end of the cannula. In these circumstances, decannulation should be accomplished as quickly as possible. Otherwise a suitably curved tube, rather short and with a smooth tucked-in

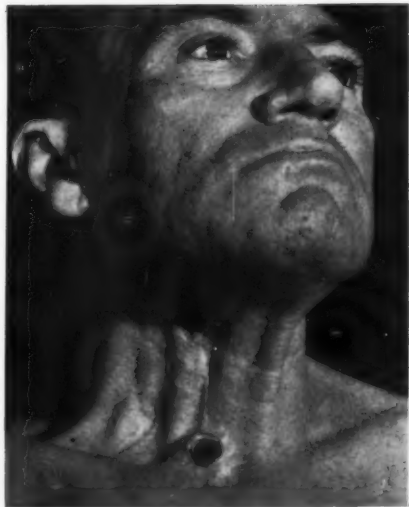
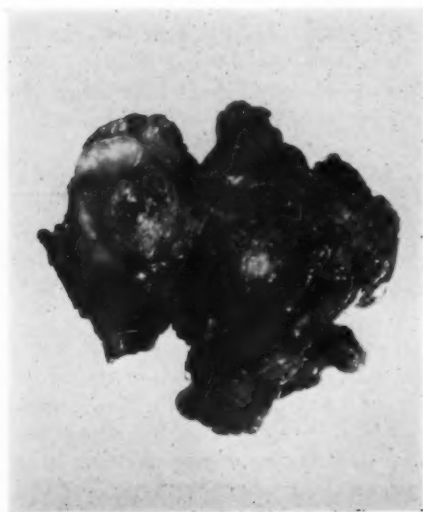


Fig. 2.—a) Large carcinoma of right pyriform sinus and extrinsic larynx, with metastasis to three of fifty nodes. b) Appearance of neck following laryngectomy, partial hypopharyngectomy and right radical neck dissection, with acrylic "button" (Moore) in situ to prevent contraction of stoma.

lower end may be helpful. Attempts to use a cannula long enough to pass beyond an already-developed stenosis may result only in the occurrence of stenosis at a still lower level. Attempts at dilatation will rarely be successful, although an indwelling hollow acrylic mold of special shape may be used.

Keloid has been satisfactorily treated in some cases by simple excision followed immediately by 1000 r of superficial x-ray irradiation in one dose. Others have used topical hyaluronidase-hydrocortisone preparations before and after excision,² or repeated applications of carbon dioxide snow.⁷

Some degree of stenosis of the hypopharynx or cervical esophagus may be anticipated when large portions of the circumference of these structures must be excised. In such cases, single-row closure of mucosa and constrictor muscle, with reliance on ribbon muscle reinforcement rather than inversion may preserve a greater lumen; the temporary fistula which may occur will not detract from the ultimate result. If only a narrow vertical strip of pharyngeal wall remains after removal of the specimen, a staged plastic repair may be required. Peroral dilatation is subsequently used as necessary, with silk-woven or rubber bougies or an esophagoscope of suitable size. The strong propulsive power of the oral cavity and pharynx may be sufficient to prevent further contracture, or actually accomplish some dilatation, when the narrowed segment is at a high level.

In laryngectomy combined with total pharyngectomy, one-stage reconstruction has been employed,^{3,9,10} although postoperative stenosis requiring frequent dilatation is said to have been a problem in spite of the use of a stent at the time of grafting. Otherwise, pharyngostoma and subsequent reconstruction is required. Martin⁷ advocates temporary closure of the pharyngostome until the last stage of repair, which is usually by means of tube pedicle grafts. The Wookey technique,¹² with sliding flaps from adjacent areas of skin, may also be applied in such cases.

The deformities and disabilities resulting from radical neck dissection, whether done in continuity with resection of larynx and adjacent structures or for subsequent metastasis, are largely unavoidable. By present standards, the procedure requires sacrifice of sensory branches of the cervical plexus (greater auricular, cutaneous colli and

supraclavicular), with resultant anesthesia and at times paresthesias of the areas supplied, and the spinal accessory nerve. The lingual nerve is usually preserved, its communication with the submaxillary gland being divided as the latter is liberated for inclusion with the specimen.

Inadvertent division of the mandibular branch of the facial nerve will result in paresis of the corresponding lower lip. Identification and preservation of this branch beneath the platysma as it crosses superficial to the external maxillary vessels a short distance below the inferior border of the mandible will avoid this deformity.

The spinal accessory nerve is divided routinely as it crosses the space between the posterior border of the sternocleidomastoid muscle and the anterior border of the trapezius. Loss of trapezius support of the scapula results in the characteristic shoulder-drop, and limitation of full lateral abduction of the arm, although anterior elevation is not greatly affected.

The hypoglossal nerve is usually preserved, often being first identified on the hyoglossus muscle as the submaxillary gland is freed. It may be followed beneath the sectioned or resected posterior belly of digastric muscle to the site of ligation of the internal jugular vein. Inadvertent division or sacrifice because of proximity to metastatic nodes will result in hemiparalysis of the tongue, but interference with mastication and swallowing is not great, unless subsequent division of the opposite hypoglossal should occur.

Unilateral section of the vagus nerve, rarely required for adequate excision of disease, is thought to be followed by no important sequelae.

Accidental division of the phrenic nerve, resulting in hemiparalysis of the diaphragm, is avoided by identification low on the surface of the scalenus anticus, beneath the crossing of the transverse cervical vessels, and careful observation as the dissection is carried upward on the deep cervical fascia covering the scalenae. The brachial plexus is readily identified as it emerges beneath the posterior border of the scalenus.

Injury or sacrifice of the cervical sympathetic chain, resulting in Horner's syndrome, is not of great importance except for the minor cosmetic effect (ptosis and enophthalmos).

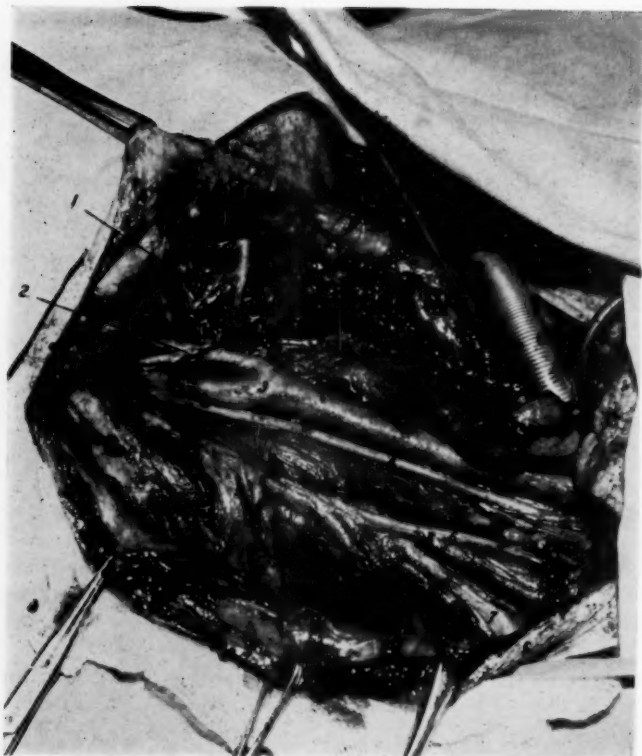


Fig. 3.—Right radical neck dissection in continuity with laryngectomy and partial hypopharyngectomy, showing structures usually preserved in neck dissection (lateral view): 1) lingual nerve, 2) hypoglossal nerve, 3) carotid artery, with internal and external branches, 4) vagus nerve, 5) cervical sympathetic trunk, 6) phrenic nerve and 7) brachial plexus.

No significant sequelae are expected as a result of unilateral jugular ligation, although as a rare complication coma and death have been attributed to interference with cerebral blood flow.^{1,2} Presumably this may occur when variations in the intracranial venous sinus pattern result in a marked predominance of jugular outflow on the operated side.

Edema and cyanosis of the face, with headache, are more frequent and more persistent sequelae following bilateral internal jugular ligation, even when an interval of several weeks or months has elapsed between sides. Simultaneous bilateral neck dissection with preservation of one internal jugular appears to be a less sound procedure than a staged operation, with an intervening period of three or four weeks prior to the second neck dissection. Martin⁶ finds that rises in spinal fluid pressure are not persistent or of serious degree following ligation of the second jugular, and that these may be due to positioning of the head or to autonomic nervous factors; he has found no increase in mortality in cases of bilateral jugular ligation.

The mortality of ligation of the common carotid artery has been thought to be as high as 50 per cent in the age group in which neck dissection is performed. Conley⁴ has listed the following possibilities in dealing with cases in which sacrifice of a segment of carotid is required. In order of choice (depending on feasibility in the individual case), these are: 1) resection and resuturing, 2) free vascular grafting, 3) ipsilateral anastomosis of external and internal carotids and 4) ligation. According to Conley, a pressure fall of 60 to 70 per cent above the point of temporary occlusion of the carotid suggests the likelihood of serious complication (hemiplegia, coma, death). Spontaneous recovery from hemiplegia following carotid ligation may occur.

Martin⁷ emphasizes the importance of avoiding hypotension at the time of, and following, carotid ligation. This precaution is reported to have accomplished a rather marked reduction in mortality.

Ligation of the thoracic duct results in no important sequel, but unrecognized injury may be followed by persisting chylous drainage requiring secondary exposure and ligation. Rarely, removal of thyroid or parathyroid in extensive procedures may require replacement therapy.

In radical neck dissection, a satisfactory cosmetic result is favored by inclusion of platysma with the skin flaps for greater viability and avoidance of, or prompt treatment of, hematoma or wound infection. Special problems in plastic repair are, of course, presented in cases where previous administration of irradiation results in delayed or incomplete wound healing.

SUMMARY

The deformities and disabilities enumerated might be considered in two groups. In the first are those which result inevitably from the nature of the surgery required in eradicating malignant disease. These include permanent tracheostomy in all cases, and in cases requiring neck dissection the deformity due to loss of the excised bloc of tissue, sensory loss (sensory branches of the cervical plexus) and loss of trapezius function (spinal accessory nerve).

In a second group might be listed the deformities which, depending on the lesion and the surgical technique, may or may not be avoided. These include stenosis of the tracheal stoma, stenosis of the hypopharynx and esophagus and pharyngostoma. Also in this group, and particularly related to the surgical technique, are paralysis of the mandibular, lingual or hypoglossal nerves.

Although adequate surgical resection is, of course, the prime objective, unnecessary sacrifice of normal tissues may be minimized by 1) complete pre-operative evaluation of the primary lesion (indirect laryngoscopy, roentgen examination, endoscopy), 2) careful identification of the structures to be preserved and 3) approach to the primary lesion through tissues known to be uninvolved, but excision under direct vision, aided by palpation, particularly in lesions involving hypopharynx, esophagus, valleculae and base of tongue.

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REFERENCES

1. Anlyan, A. J., Browning, H. C., Black, S. P. W., Clifton, E. E., and Janzen, A. D.: Dural Sinus Studies in Relation to Radical Operations of the Neck. *Surgical Forum, Clin. Cong. Am. Coll. Surg.*, pp. 300-305, 1951.
2. Belisario, J. C.: The Treatment of Keloids. *Acta Dermat-Venereol.* 37:165-181, 1957.

3. Conley, J. J.: One-Stage Radical Resection of Cervical Esophagus, Larynx, Pharynx and Lateral Neck, with Immediate Reconstruction. *Arch. Otolaryng.* 58: 645-654 (Dec.) 1953.
4. Conley, J. J.: Carotid Artery Surgery in the Treatment of Tumors of the Neck. *Arch. Otolaryng.* 65:437-446 (May) 1957.
5. Gius, J. A., and Grier, D. H.: Venous Adaptation Following Bilateral Radical Neck Dissection with Excision of the Jugular Veins. *Surgery* 28:305-321 (Aug.) 1950.
6. Martin, H., DelValle, B., Ehrlich, H., and Cahan, W. G.: Neck Dissection. *Cancer* 4:441-499 (May) 1951.
7. Martin, H.: Surgery of Head and Neck Tumors. Paul B. Hoeber, Inc., New York, 1957.
8. Moore, E.: Plastic Tracheostomic Button. *J.A.M.A.* 165:1276-1277 (Nov. 9) 1957.
9. Negus, V. E.: Reconstruction of the Pharynx after Pharyngo-esophagolaryngectomy. *Brit. J. Plastic Surg.* 6:99-101 (July) 1953.
10. Shaw, H. J., and Ormerod, F. C.: Pharyngolaryngectomy with Primary "Sleeve-Graft" Reconstruction. *J. Laryngol.* 71:175-201 (Mar.) 1957.
11. Sugarbaker, E. D., and Wiley, H. M.: Intracranial Pressure Studies Incident to Resection of the Internal Jugular Veins. *Cancer* 4:242-250 (Mar.) 1951.
12. Wookey, H.: Surgical Treatment of Carcinoma of the Hypopharynx and Esophagus. *Brit. J. Surg.* 35:249-266 (Jan.) 1948.

XLI

REHABILITATION OF THE POST-LARYNGECTOMIZED PATIENT

THE VOCAL THERAPIST: PLACE AND CONTRIBUTION
TO THE REHABILITATION PROGRAM

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The problem of the voice of the laryngectomized patient has been much discussed, but the part of the voice therapist in the rehabilitation of the patient has never been outlined sufficiently. In the postsurgical therapeutic procedures, the surgeon watches the organic condition and hopes for the best when the patient is provided with voice therapy. Yet just this postsurgical phase needs the care of the physician instead of that of nonmedical persons.

It has been traditionally accepted that esophageal speech is produced by the swallowing of air, followed by reversed peristalsis with the resultant belches then formed into esthetically acceptable voice and speech. The need for psychological guidance has long been recognized, both because of the comparatively high percentage of suicidal tendencies before and after laryngectomy, and of the failure of many patients to attend special rehabilitative classes for any length of time. It is recognized that something must be done to "cheer and encourage the patient." These words are quoted from a folder published for an American Medical Association exhibit on rehabilitation of the laryngectomized.¹

Is this attitude realistic? Should the voice therapist "cheer and encourage" or should one find out what actually disturbs the patient? Is the task fulfilled by teaching a patient to belch esthetically? If eventually he can transform belches into understandable sentences, is that enough?

It has been suggested for several decades to start voice therapy before surgery.² In trying to insist upon this step, I had little success,

mostly for technical reasons. Introducing a successful laryngectomized speaker to a candidate for laryngectomy might relieve him from some fears but can give him no idea what loss of voice means. The use in everyday language of the term, "he remains speechless," indicates panic or terror, a feeling not to be prevented by reassuring the patient that later he will learn to talk. The part his personality plays and an assessment of his inner resources can seldom be made under the emergency of surgery for cancer.

What communication actually means to a specific individual is not known to the surgeon. Any form of neurosis can to some extent be explained as a disturbance in the area of transmission of messages to others.³ If therefore a neurotic suffers from cancer of the larynx the mechanical part of oral communication will be added to the psychological difficulty in transmitting messages. The resulting emotional collapse often cannot be anticipated since previously the patient might have well compensated for his neurotic difficulty. Here the strength and quantity of inner resources are of decisive influence.

To accept a situation is the result of generally successful adjustment, for which vocal analysis of the normal voice can be used as an excellent test.⁴ The balance between melody and rhythm, between head- and chest-register, the satisfactory placement within the potential range, are vocal dynamics which according to my research on voice and personality express this homeostasis.⁵ These elements, all completely destroyed by laryngectomy, cannot be brought back by even the most successful vocal rehabilitation. A completely new type of vocal dynamics will appear, without any biological rhythm, with limited range, limited melodious qualities. Registers, most delicate functions within certain fibers of vocal cords, cease to exist. Melisms, the finest expressions of personal appeal, which like a hairline brush touch in a painting can give minute variations to the meaning of messages, can never be accomplished again. We know that when we talk tongue in cheek, when we express bluffing or irony with the shortest pauses, the smallest glidings in pitch, the tiniest, hardly measurable inflections, these most personal, most individual expressions are lost with the written or printed words. They must be heard to be understood. This hurdle the laryngectomized speaker can hardly ever take. He regresses to a vocal status of childhood when only undifferentiated basic sounds were heard. He is unable to express his abstract thinking in vocal abstractions.

Laryngectomy voice, even at its best, cannot convey messages beyond rational content. Contrary to vocal expression of children before speech starts, it can hardly express pleasure or displeasure, since the difference between hard and soft attack is missing. There is no choice between the wide range of excitement and narrow range of depression. It is to normal voice as printed words are to spoken ones. The true underlying meaning therefore has to be interpreted additionally by manual gestures and facial expression.

It is obvious that the loss of the larynx is related to the feeling and fear of castration. The larynx is a secondary sex organ: most of the physiological vocal changes are secondary sex symptoms. The feeling of being emasculated concerns men only; the term, "Adam's apple," expresses the close sexual relation of the larynx. According to folk physiology the body of the first man was wiser than his soul; when Adams took from Eve his piece of the forbidden fruit, a bit of it stuck in his throat, the lump now called the Adam's apple.⁶ As a sex symbol its loss comes close to castration feelings. Whenever we observe how much easier female laryngectomized patients learn to talk and how much faster they usually adjust themselves to their condition, we see how little this complex interferes with their adjustment. The Adam's apple starts protruding during puberty, when disturbed mutation becomes a frequent and marked neurotic symptom in boys, while in girls it hardly plays a significant part. On the other hand girls usually start talking earlier and the proportion between stuttering boys and girls is five to one!

The patient who suffers from any type of carcinoma will thereafter be afraid of metastasis, however successful the surgery. The emotional condition can become an anxiety neurosis. In addition, the laryngectomized patient suffers from the inability to communicate successfully beyond rational expression. He as a personality can no longer use voice for his most personal expression; the resulting frustration disturbs further the emotional homeostasis.

We know that every type of neurosis is expressed through a specific vocal syndrome, that the audible voice of neurosis often produces visible symptoms. The organicity is secondary to emotional difficulties, a psychosomatic problem. If the voice produced by vocal cords reveals these symptoms, it goes without saying that one hears as well the pseudovoice of neurosis, produced by the mouth of the

esophagus. When allergies, the result of emotional imbalances, produce hoarseness due to swelling of the cords, one has to understand that mucous membranes of the trachea will become the seat of allergic disturbances, since the choice of the allergic organ can be a matter of conversion, of the translation of emotional disturbances into organic ones. This explains the frequent vocal difficulties of the laryngectomized patient because of excessive mucus, dryness, spasms of the esophageal wall (which are visible in x-rays),⁷ the symptoms of anxiety and frustration of disturbed communication. In addition, the initial loss of smell and the frequently disturbed defecation caused by lost glottis pressure interfere with the well-being.

TECHNIQUE OF ESOPHAGEAL VOICE

The principle of an up-to-date therapy can be condensed in the idea: it is neither air-swallowing nor belching which will produce an esthetically acceptable voice. It has been found that the first step in swallowing consists of lifting the esophageal sphincter and retracting the tongue. The oral cavity is closed and air is injected from the pharynx into the esophagus. The phases of deglutition are entirely unnecessary to produce voice. The plosives, *p, t, k, d*, can often be produced immediately. By this method esophageal pressure is independent from expiration, and less noise from the tracheostoma interferes with voice. Experiments have shown that the syllable, *PA*, could be repeated 40 times with one intake of air into the esophagus, while a new breath had to be taken about every 15 seconds.⁸ If the plosives do not serve the purpose fast enough, I start with syllables like "*sky*" which help to bring the tongue into proper position.

Anatomically speaking, it is well known that the condition of the glossopharyngeus muscle influences the prognosis of vocal therapy. However, the injection method proves that even if the muscle is in poor condition one still gets a good result in vocal rehabilitation. The formation of the pseudoglottis and its changes can be well observed in stroboscopic x-ray pictures, and even better with movie x-rays: Whatever method is used the clinician needs the control over possible difficulties in placing the pseudoglottis or recognizing the very frequent spasms and functional changes within the esophageal region.

WHO SHOULD PROVIDE VOICE THERAPY?

The physiological, psychological aspects as well as the x-ray control prove that medical help is absolutely necessary. The function

of the surgeon is by no means limited to laryngectomy, postsurgical control of the tracheostoma and recheck for possible metastasis. He who has taken away the voice is logically the responsible person for the restoring of this function. He has both the responsibility and the authority; he has become the healing father figure, and the patient trusts him more than he does anybody else. If he lacks the time to give vocal therapy he should supervise it regularly instead of being satisfied that his patient enrolls in a class. Specialists who would never allow the registered nurse to wash the ear of a patient often do not hesitate to entrust speech therapy to a layman's care.

The most unrealistic method is to hire a vocally successful laryngectomized patient as a teacher for groups. In rare cases this has been a success. One would have to know that this teacher is emotionally and technically equipped for this task, and not just an overcompensating "professor." The problems of the patient are so complicated that a lay person can help only the few balanced ones. I have observed very often how previously compensated emotional tendencies in a patient become activated through laryngectomy and how he escapes into neurosis. Schizoid tendencies can become marked schizophrenic ones. Voice teachers always demonstrate their successful patients. One hears nothing about the ones who do not learn, buy an artificial larynx or drop out of the courses. The association with a fellow-sufferer as teacher is by no means always a therapeutic factor. It is naive to compare teaching classes for laryngectomees with group therapy in psychiatry. The voice therapist needs the psychological training and the insight to recognize the attitudes of his patients toward communication. A few will always commit symbolic suicide: death and silence are twins. Those who do not speak express their wish to be dead.

The ideal treatment will be through the joined forces of surgeons and medical voice therapists who are able to do more than just teach the patient how to belch esthetically. They must develop new methods for therapy and control by gaining deeper insight into the patient's problems by careful observation and diagnosis.

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REFERENCES

1. McDonald, Eugene T.: The Rehabilitation of the Laryngectomized. Am. Med. Assn., Scientific Exhibit, 1946.
2. Moses, Paul J.: Speech and Voice Therapy in Otolaryngology. Eye, Ear, Nose and Throat Monthly 32:367-375, 1953.
3. Ruesch, Jurgen, and Bateson, Gregory: Communication. Norton and Co., New York, 1951.
4. Moses, Paul J.: Vocal Analysis. Arch. Otolaryngol. 48:171-186, 1948.
5. Moses, Paul J.: The Voice of Neurosis. Grune and Stratton, New York, 1954.
6. Shipley, Joseph T.: Dictionary of Word Origins. Philosophical Libr., N. Y., 1951.
7. Brankel, Otto: Formung und Gestalt der Pseudoglottis Laryngektomierter im Stroboskopischen Röntgenbild. Folia Phoniatica 9:13, 1957.
8. Van den Berg, Jw., Moolenaar-Bijl, A. J., and Damsté, P. H.: Esophageal Speech. Folia Phoniatica 12:2, 1958.

XLII

REHABILITATION OF THE POSTLARYNGECTOMIZED PATIENT

SPECIFIC DISCUSSION OF FAILURES ADVANCED AND DIFFICULT TECHNICAL PROBLEMS

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Since the voice following laryngectomy is dependent upon a chamber for the storing of air and a vibrating mechanism by which sound is produced on expulsion of the trapped air, failure to develop a voice may be due in a limited number of cases to extensive technical procedures which encroach upon both these factors. Pharyngeal or upper esophageal stenosis is a complication in future speech training, far more than lower esophageal disturbances. The inferior constrictor muscle has been thought to be a factor in voice production but when the cricoid cartilage is removed in laryngectomy it takes away the lower attachment so that there is no fixed point upon which the muscle can act, although it may have some importance in establishing a vibrating mechanism. This upper esophageal sphincter with its striated muscle fibers is partly under voluntary control and can be trained to both inspire and expire air, at the same time establishing enough contact with the surrounding mucosal tissue to cause vibration. While failure of the membrane in the pharynx to vibrate either due to extensive scar tissue or fixation may hinder the creation of a buccal voice there is little evidence to incriminate anatomical factors as the primary cause for lack of voice.

Many patients have adequate reservoirs and resonating mechanisms, yet will only say one or two indistinct words. Some contend that they cannot learn to swallow air while others are unable to use the swallowed air for more than several syllables. A few inspire deeply and sharply through the tracheal stoma at each attempt to speak so that their voice is clouded by the inspiratory noise in the trachea. Still other patients have complained of severe heartburn

and epigastric pain on swallowing air and have discontinued attempts to talk. Air need not enter the stomach and can be stored either in the pharynx or esophagus so that there is little basis for the latter complaint unless the patient swallows large gulps of air into the stomach without regurgitation. Failure to talk satisfactorily for these reasons is not due to anatomic limitations imposed by advanced surgery but can be laid to the patient's lack of energy, fortitude or industry necessary to learn any new method.

In order to determine what factors if any had a bearing upon failure to develop a voice from an anatomical or physiological standpoint following laryngectomy an analysis was made of 440 patients operated upon at Jefferson Hospital (Table I). One hundred sixty-six

TABLE I
VOICE AFTER LARYNGECTOMY

Useful buccal voice	274	62%
No voice	141 (32%)	
Artificial larynx	25 (5.7%)	38%
Total	440	100%

patients (38 per cent) failed to develop a useful buccal voice. Included in this number were 25 who had a voice but felt that the voice was not distinct or serviceable. Only 25 patients (5.7 per cent) used an artificial larynx. The percentage of failures in this group was higher than that reported by Heaver and his associates¹ on 274 laryngectomized patients, in which 27 per cent did not develop a voice and 4.4 per cent used an electrolarynx. Most of the patients with a buccal voice had an excellent method of communication and once the initial technique was mastered the voice became useful and well controlled. Overcoming the difficulties in learning the new technique seemed to be the important step in the production of a voice and once developed it proved to be quite satisfactory.

Men far outnumbered women as expected and they also were more adept at learning to talk. Among the males, failures occurred in approximately one-third, while of the 23 women approximately

two-thirds were unable to talk. There is no apparent difference in the basic musculature of the male and female pharynx to account for this reversal of percentages, but psychologic elements associated with fixed emotional attitudes were strong factors in the women and generally deterred them from perseverance. Many of the women disliked being the center of attraction and considered that a buccal voice made them conspicuous. When people turned to see who was talking with a unique, deep throaty sound expecting to see a man and found a woman it sometimes caused embarrassment. Some women thought they were less conspicuous with no voice at all, while others failed to develop a voice because of an over-sensitive esthetic sense.

In reviewing the 166 cases (38 per cent) that did not develop a useful voice no constant underlying physical reason was found. The vertical, Y-shaped, H-shaped and collar incisions were variously employed with no appreciable difference. There were patients who had a classical laryngectomy with primary closure of the pharynx re-enforced by a second layer of inferior constrictor muscle while others had removal of most of the musculature, pharyngeal wall and contiguous structures of the neck. Stenosis with narrowing of the air space and marked fibrosis was nearly always associated with pharyngectomy but, since it is not necessary to have a large reservoir for air in order to speak, the reduced air space from extensive surgical removal did not particularly interfere with the establishment of a voice. When the entire mucous membrane together with the underlying supporting musculature was removed voice production was not materially affected provided primary anastomosis between the base of the tongue and the remaining pharynx was accomplished. As many patients with extensive surgical removal developed a voice as did those with conservation of the pharyngeal muscles, so that anatomical and physiological tissue loss in the pharynx, hypopharynx or cervical esophagus had little significance.

Routine removal of either the entire or the central portion of the hyoid bone did not alter the postlaryngectomized voice. Without this upper attachment the strap muscles of the neck were useless as far as function was concerned and were removed with the larynx. At times a rather firm shelf or fibrous band formed in the pharynx at the previous site of the epiglottis and, although it occasionally caused lodgement of food, it had no significant bearing on the voice. In patients who had unilateral or bilateral neck dissections combined

with laryngectomy no interference with the buccal voice was noticed, which substantiates the belief that the neck muscles are not necessary to expel the trapped air in order to produce a sound. Their only function is to splint the neck. The patient's life should not be compromised when doing a laryngectomy by leaving behind the strap muscles, epiglottis or hyoid bone since their retention may lead to subsequent development of malignancy and their removal does not increase the patient's disability nor interfere with the development of a buccal voice.

The matter of closure of the pharynx should be considered, for at one time it was thought that the buccal voice was dependent upon the cricopharyngeal bar. Attempts have been made by some to repair and establish a new cricopharyngeus muscle when it was interfered with by surgery. These attempts met with little success and the patients in whom the entire cricopharyngeus muscle was removed without reconstruction presented voices as satisfactory as those in whom it had been repaired. Currently I am using the horizontal pharyngeal closure but only because it is felt that the blood supply is better preserved and the healing occurs more rapidly. No difference in the voice has been noticed regardless of the manner of closure. The only benefit that might be derived from a serviceable and highly developed cricopharyngeal and inferior constrictor muscle is to maintain the swallowed air better under the voluntary control of the patient.

For phonation it is necessary to move the tongue, particularly the base, and touch the tongue against the hard palate. In advanced disease when the hypoglossal nerve was sacrificed on both sides the patients not only experienced difficulty in swallowing food but also in establishing a voice. Operations which impeded tongue movement by removal of a portion of its base hindered voice production to a certain degree, but the patients were able to speak and the only abnormality noted was failure to say more than two or three words with one gulp due perhaps to inability to force air downward and gradually expel it because of lack of constriction at the upper aperture. The volume of the sound was excellent. Limitation of the motion of the tongue, preventing the intake of air into the pharynx also proved to be a deterring factor. Disturbance or removal of the musculature in the floor of the mouth particularly the mylohyoid muscle presented difficulties in initiating the swallowing act. Excision

of the hyoid bone removed the anchorage of the muscles supporting the floor of the mouth as well as the strap muscles. The tongue and the underlying muscles in the floor of the mouth are probably the single most important element in initiating the act of air swallowing, and disturbances in their function interfered with early attempts to learn to speak, but once mastered the voice was just as proficient as that produced when these structures were intact.

In the surgical treatment of patients with carcinoma of the larynx complete removal of the malignancy should be carried out without regard to the subsequent establishment of a voice. Adjacent structures which are not necessary to carry on life and which do not lead to disability when removed can be sacrificed, enhancing the likelihood of eradicating undetected malignancy. Development of a voice can be accomplished no matter whether the larynx is removed with no contiguous tissue or whether the larynx and the surrounding structures are removed. In some patients difficulty in the production of voice after removal of the larynx leaving only the skin and platysma was anticipated, but in these patients the voice has been just as satisfactory as in those in which the inferior constrictor muscle was preserved and used to re-enforce the pharyngeal closure. In advanced cases where the retained pharyngeal mucosa was limited to several millimeters and it was necessary to close the greater part of the pharynx over a feeding tube to secure a lumen, subsequent healing resulted in no deterrent to the development of a voice.

In performing cancer surgery one should not be planning for the future as far as the voice is concerned for, if this thought is present in the mind of the surgeon, he may forget that the primary object in operating is to extirpate the carcinoma. Cancer may not only spread to the lymph nodes of the neck, but it may continue into the muscle sheaths, penetrate the epiglottis invading the pre-epiglottic space, the base of the tongue and the hyoid bone without being visible or palpable on examination of the neck prior to operation. Leaving behind any of these structures in a conservative approach may result in an incomplete operation, and when one attempts to conserve tissue no matter for what reason the chances of a successful cure are diminished and eventually everything becomes lost. Recurrence of malignancy in the neck other than in the lymph nodes means an incomplete operation indicating that the primary surgery was not thorough and the removal not adequate. When one begins to speculate about leav-

ing this or that behind in order to give the patient a better voice more recurrences before five years have elapsed will develop than if the malignancy was treated and no one worried about speech at the time of the surgery.

SUMMARY

In a survey of 440 laryngectomized patients 166 (38 per cent) failed to develop a useful buccal voice and of this group 25 (5.7 per cent) used an artificial larynx. Males proved more adept than females in developing a voice in the ratio of 2 : 1. No underlying anatomic or physiologic factor was found to account for the lack of voice, and patients with extensive surgical removal including most of the pharynx and portions of the tongue as well as the larynx were able to talk expertly. The type of neck incision, method of pharyngeal closure, or amount of adjacent cervical tissue excised had no appreciable effect upon the establishment of a voice.

The one anatomical factor of minor significance in the production of a voice after laryngectomy was disturbance of the musculature of the tongue and floor of the mouth, for unimpeded motion of these structures was necessary both to initiate the act of swallowing and also to phonate distinctly. While the technique of swallowing air could be learned the voice was not as well controlled in patients in whom portions of the tongue were removed.

Since the primary object in the surgical treatment of carcinoma of the larynx is complete eradication of the malignancy this should be carried out regardless of the sacrifice of adjacent tissues and heedless of the subsequent development of a voice, which can be accomplished equally well following advanced and difficult technical procedures as after uncomplicated classical laryngectomy once the initial technique has been mastered.

1712 LOCUST STREET

REFERENCES

1. Heaver, L., White, W., and Goldstein, N.: Clinical Experience in Restoring Oral Communication to 274 Laryngectomized Patients by Esophageal Voice. *J. Am. Geriatrics Soc.* 3:687 (Sept.) 1955.

XLIII

PSYCHOLOGICAL FACTORS DETERMINING THE SUCCESS OR FAILURE OF THE REHABILITATION PROGRAM OF LARYNGECTOMIZED PATIENTS

BERNARD STOLL, M.A. (by invitation)

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The concern for the rehabilitation of the patient who has experienced a laryngectomy probably dates back to 1887 when Stoerk¹ observed that one of his patients developed a pseudo-voice following total laryngectomy due to cancer. Since 1887 more efficient methods of early cancer detection have been devised, and the laryngectomy operation has been used more extensively and successfully. The resultant increase in longevity has made it mandatory that the laryngectomized patient find his rightful place in society. This paper will attempt to investigate some of the factors which might contribute to the success or failure of the rehabilitation program of the laryngectomized patient.

A review of the controlled research conducted in this area reveals a paucity of information relative to the rehabilitation problem involved. Investigators have concerned themselves primarily with describing what is happening both physiologically and acoustically when a person without a larynx uses esophageal speech in his attempt to communicate with and thus adjust to society. Stern,² Morrison,³ Brighton and Boone,⁴ Stetson,⁵ Burger and Kaiser,⁶ Amster,⁷ Yellow Robe,⁸ Seeman,⁹ Bateman, Dornhorst and Leathert,¹⁰ Negus,¹¹ Anderson,¹² Hyman¹³ are some of the investigators whose research interest was the examination of the functions of the structures involved in the production of esophageal speech. Amster,⁷ Anderson,¹² and Hyman¹³ have shown that the proficiency of esophageal speech varies from speaker to speaker and that standard speech intelligibility indexes can objectify this variation.

The aforementioned investigators have given us most of the research information to which we can refer. It is often impossible to

gain access to enough laryngectomized patients under controlled situations to conduct such research.

On the other hand, much information pertinent to the rehabilitation problem comes to us from the clinical observations of physicians and speech pathologists. Schall¹⁴ was one of the first physicians to indicate that the mental outlook of the patient should be given serious consideration when a laryngectomy is contemplated. He writes as follows: "The introvert who has difficulty in facing any crisis is usually very poor at making the mental readjustment necessary after total laryngectomy." Levin¹⁵ reports that: "Emotional disturbances of varying degrees occur in nearly all patients. While the introverted person suffers more in proportion, even the extrovert is depressed when he finds himself entirely speechless." Greene¹⁶ states that the individual's interpersonal relations with physicians, nurses, family and friends impede or hasten rehabilitation. Beamer¹⁷ in her study of the personality adjustments of laryngectomized subjects observed that: "An experience of the traumatic intensity of a laryngectomy by its very nature has the potential of a disturbing effect upon the individual's sense of belonging, his self-concept, his attitudes, and many other aspects of his phenomenological field." Her research indicated that although no M.M.P.I. profile was found to be typical of laryngectomized subjects, seven of a total of eight profiles were deviant.

We can logically expect certain temporary personality changes to result from a total laryngectomy experience. The causes of such personality changes if these changes are indeed present can be attributed to the anxiety evoked in the presence of certain fears associated with the entire laryngectomy experience.

The fears often experienced by patients prior to the laryngectomy are as follows: 1) The fear of the word cancer and the many semantic implications involved in such a word. The fear of death is probably the paramount fear. 2) The fear of operations in general. 3) The fear of the permanent loss of voice. The anxiety associated with this fear results from the assumed consequences of permanent aphonia. The patient's entire pattern of interpersonal relationships built up through the years becomes threatened. He worries about the probable loss of his job, his security, his friends, etc.

The fears often experienced by patients postoperatively are as follows: 1) The fear of the recurrence of the cancer, hence the con-

tinued fear of death. 2) Fears due to the new physiological relationships resulting from the laryngectomy. (The inability to lift heavy objects, the breathing and coughing from the tracheal stoma, the often impaired sense of smell and taste, the cosmetic liabilities of the tracheal stoma, etc.) 3) The fear of old age which has been aggravated by the feeling of uselessness resulting from the loss of speech. The depression frequently observed often has its roots in this specific fear. The loss of earning power further contributes to this feeling of uselessness. 4) Fear of being unable to re-establish old patterns of interpersonal relationships. 5) The fears associated with the anticipation of failing to learn a new method of speaking.

In order to eliminate as many of the above sources of anxiety as is possible, the rehabilitation program should begin with intensive counselling. Pre-operatively the burden for this counselling falls upon the shoulders of the first physician diagnosing the condition. Many of the patient's pre-operative fears can be removed or at least reduced by such action. The presence of an intelligent, psychologically astute person who has experienced a laryngectomy and has learned to use good esophageal speech can be of great support to the patient at this time. The consultant speech pathologist can assist the physician with this basic counselling problem.

Postoperatively the counselling program should move into the hands of the speech therapist. The therapist, if possible, should help to organize a group of laryngectomy patients. Although actual speech retraining is conducted on an individual basis, within the framework of group therapy most of the fears mentioned above can be discussed openly. The support thus gained is of immeasurable help to the newly laryngectomized patient.

Realistically, however, the fears resulting specifically from the lack of speech can be removed only by the acquisition of a new speech pattern. The rehabilitation program is entirely dependent upon such acquisition. The new speech pattern referred to will be that of esophageal speech. It will not be within the scope of this paper to discuss the advantages or disadvantages of the speech of a mechanical or electrical larynx. This writer agrees with Kallen¹⁸ when he says: "For psychological reasons the mastery of vicarious voice is of great importance in maintaining the health of the laryngectomized person's psyche. A mechanical device can never have the same psychological

significance as a living organ with a newly developed function. Such a function becomes entirely identified with personality. This is not likely when a mechanical device is used."

For many years speech pathologists have been aware of the fact that, although most laryngectomized patients can learn esophageal speech, the quality of the esophageal speech learned varies from person to person. A study conducted by this writer¹⁹ found that society's attitude toward and acceptance of esophageal speech was correlated significantly with the objective measurements of the intelligibility of this same esophageal speech. If society will accept only the more intelligible esophageal speech, then it becomes urgent that the factors preventing the learning of such speech be determined.

Research to date has been unable to arrive at any physiological reason for such variation of intelligibility. It has been this writer's clinical observation that the chief factor responsible for this variation has been the motivation with which the laryngectomized patient approaches the learning of more refined esophageal speech patterns. The attitude that the patient has toward verbal communication is indicative of this motivation. Knower²⁰ supports this hypothesis by stating that in the learning of any speech pattern "a favorable attitude or 'set' toward speech indicates a predisposition to speak and reflects an interest which should broaden experiences through which learning may take place. The favorable attitude may itself facilitate learning." Other authors have stressed the importance of considering those factors which might affect the motivation of the laryngectomized patient. Levin²¹ writes as follows: "Necessity and willingness to learn are the essential factors. This is exemplified by a patient aged 29 who was compelled to learn to talk, having the responsibility of a family and the necessity of earning a livelihood. He attacked the problem aggressively, learned readily and achieved excellent results." Morrison,³ and Brighton and Boone⁴ agree when they say that "perseverance in practice" and "the amount of effort expended in learning voluntary control of an involuntary speech mechanism" are essential requirements for the mastery of esophageal speech. To achieve such perseverance, the patient must be highly motivated to learn esophageal speech. Greene²² and Faulkner²³ indicate the physiological basis for assuming a relationship between attitudes and quality of esophageal speech when they report that spasms of the esophagus can be increased and the lumen narrowed by suggestions which arouse such emotions

as grief, anger, anxiety, fear and apprehension. It was noted that a relaxation of the spasms and a widening of the lumen occur under suggestions which arouse emotions such as happiness, elation, contentment, security and enthusiasm.

In another study,¹⁹ this writer has determined that the laryngectomized patients having the most intelligible esophageal speech were those who scored highest on a test designed to measure the healthiness of their speech attitudes. Another study, conducted by this writer,¹⁹ revealed that speech experience inventories determining the amount of speaking done during the patient's pre-operative life was significantly related to the intelligibility of the esophageal speech which he later learned. It seems probable that to the degree that the patients were used to expressing themselves verbally before the laryngectomy, to that degree were they motivated to learn the more refined patterns of esophageal speech necessary to continue this verbal behavior pattern.

Some of the factors which might prevent a patient from achieving the motivation necessary for learning more intelligible esophageal speech are as follows: 1) the personality of the patient; 2) lack of an opportunity for speech therapy; 3) the patient's over-concern of the attitudes of society toward esophageal speech.

Many clinicians have observed that it is easier to motivate certain personality types. Levin²¹ comments upon the mental attitude of laryngectomized patients as follows: "The aggressive extrovert goes after the problem of learning in a direct and businesslike way and acquires the method rather easily: he has the attitude of the good student. The emotionally unstable and introverted type, not having fully recovered from the psychic trauma incidental to the diagnosis of cancer and the subsequent radical operation and hospitalization, finds it difficult to concentrate and to learn anything new."

Schall¹⁴ reports that "the extrovert makes his readjustment most readily and takes great pride in his accomplishment." Pitkin's²⁵ reference to the "mature, out-going person" reflects his agreement with the above authors that the extroverted patient is more highly motivated and will probably be more successful in learning superior esophageal speech.

It is indeed unfortunate that in many areas of the country adequate speech rehabilitation services for laryngectomized patients are unavailable, and the physician is often too busy for adequate personal follow-up. (The Executive Secretary of the American Speech and Hearing Association, 1001 Conn. Avenue N.W., Washington 6, D.C., will be glad to forward the name of the nearest competent speech and hearing therapist.)

The patient's evaluation or misevaluation of the attitude of society toward his esophageal speech is often responsible for reduced motivation to learn more precise esophageal speech patterns. He has often, unfortunately, been told that the production of esophageal speech is based on a physiological principle similar to that of eructation or belching. Many of the earlier investigators used the word belch in their description of this process. Stetson⁵ writes: "The subject gulps a large amount of air, retains it probably in the stomach, and speaks his phrase on one long, hasty, belching breath." To the patient thus conditioned almost all of the initial esophageal speech attempts seem to have acoustic properties similar to the sound of belching. That the process of belching has certain unpleasant connotations is undeniable. Baker²⁴ seems aware of this aspect of the problem when he says that the patient must be given guidance in the adjustments he must make to free himself from the fears, embarrassments and insecurities that attend public recognition of his disability. Anderson,¹² Hyman,¹³ and Amster⁷ hint at the importance of the role that society must play in order to obtain a realistic evaluation of esophageal speech as a method of communication.

The studies conducted by Stoll¹⁰ reveal that poor intelligibility rather than the low pitch associated with belching is responsible for certain negative attitudes toward esophageal speech. The patient can learn to use esophageal speech without ever being exposed to the word belch. A host of misevaluations can thus be avoided, and the patient can be motivated to work harder at establishing finer esophageal speech patterns. He must also learn to properly evaluate society's reaction to his speech. If the patient feels his speech is not being accepted by society, he will reduce the number of speaking contacts he has with society. If he reduces the number of speaking contacts he has with society, he will not have enough practice to enable him to achieve more intelligible esophageal speech, and if he does not achieve more intelligible esophageal speech, society will not accept

him. The therapist must break this cycle by wisely counselling the patient and by setting goals high enough so that the resulting esophageal speech will be accepted by society. It is in this area that additional research is sorely needed.

FRESNO STATE COLLEGE

REFERENCES

1. Stoerk, K.: Ueber Larynxextirpation. *Wien. Med. Wchnschr.* 37:1535, 1887, cited by Morrison, W. W.: The Production of Voice and Speech Following Total Laryngectomy. *Arch. Otolaryng.* 14 (Oct.) 1931.
2. Stern, Hugo: Grundprizipien der Sprache und Stimmbildung bei Laryngektomierten nebst einem neuen Beitrag zum Mechanismus des Sprache und Stimme derartig Operierter. *Wien. Klin. Wchnschr.* 33:540, 1920, cited by Kallen, L. A.: Vicarious Vocal Mechanisms. *Arch. Otolaryng.* 36 (July-Dec.) 1937.
3. Morrison, W. W.: The Production of Voice and Speech Following Total Laryngectomy. *Arch. Otolaryng.* 14:413-431 (Oct.) 1931.
4. Brighton, G. R., and Boone, W. H.: Roentgenographic Demonstration of Method of Speech in Cases of Complete Laryngectomy. *Amer. Jour. Roentgen. and Rad. Ther.* 38:571-583 (July-Dec.) 1937.
5. Stetson, R. H.: Esophageal Speech for Any Laryngectomized Patient. *Arch. Otolaryng.* 26:132-142 (July-Dec.) 1937.
6. Burger, H., and Kaiser, L.: Speech Without a Larynx. *Acta Otolaryngologica* 8:90-116, 1925.
7. Amster, W. W., Di Carolo, L., and Herer, G.: *Speech After Laryngectomy.* Syracuse University Press, 1955.
8. Yellow Robe, E. M.: A Study of the Role of Three Factors in the Development of Speech After Laryngectomy: Type of Operation, Site of Pseudoglottis, and Coordination of Speech with Respiration. Unpublished Phd. Dissertation, Chicago: Northwestern University, 1954. Mic A 54-2737.
9. Seeman, M.: Phoniatische Bemerkungen zur Laryngektomie. *Archiv. fur Klinische Ghirurgie* 140:285-298, 1926.
10. Bateman, B. M., Dornhorst, A. C., and Leathart, M. B.: Esophageal Speech. *Brit. Med. Jour.* 2:1177-1178 (Nov. 29) 1952.
11. Stetson, R. H.: Esophageal Speech: Methods of Instruction After Laryngectomy. *Arch. Neerlandaises de Phonetique Exper.* 13:95-110, 1937.
12. Anderson, John O. Dean: Study of Some Factors Concerning Esophageal Speech. Unpublished Doctoral dissertation, Ohio State Univ., 1951.
13. Hyman, M.: An Experimental Study of the Relative Pressure, Duration, Intelligibility and Esthetic Aspects of the Speech of Artificial Larynx, Esophageal, and Normal Speakers. Unpublished Ph.D. dissertation, Ohio State University, 1953, p. 129.

14. Schall, L. A.: Psychology of Laryngectomized Patients. *Arch. Otolaryng.* 28:581-584 (Oct.) 1938.
15. Levin, N. M.: Speech Rehabilitation After Total Removal of the Larynx. *Jour. A.M.A.* 140:1281-1286 (Aug.) 1952.
16. Greene, J. S.: Speech Rehabilitation Following Laryngectomy. *Amer. Jour. Nursing* 49:1-2, 1949.
17. Beamer, M. W.: A Qualitative Study of Personality Adjustment of Laryngectomized Subjects. Unpublished Master's thesis, Texas St. College for Women (June) 1954.
18. Kallen, L. A.: Vicarious Vocal Mechanisms. *Arch. Otolaryng.* 20:460-503 (July-Dec.) 1934.
19. Stoll, Bernard: Unpublished study sponsored by a federal grant from the Office of Vocational Rehabilitation.
20. Knower, F. H.: A Study of Speech Attitudes and Adjustments. *Speech Monographs* 5:1:130-203, 1938
21. See Number 15.
22. Greene, J. S.: See number 16.
23. Faulkner, W. B., Jr.: Objective Esophageal Changes Due to Psychic Factors. An Esophagoscope Study with a Report of 13 Cases. *Amer. Jour. Med. Sci.*, pp. 796-803, 1940.
24. Baker, H. K.: The Rehabilitation of the Laryngectomized. *Trans. Am. Acad. Ophthal. Otolaryngol.* 52:227-233, 1947-1948.
25. Pitkin, Y. N.: Factors Affecting Psychologic Adjustment in Laryngectomized Patients. *A.M.A. Arch. Otolaryngology*, pp. 38-49 (July) 1953.

XLIV

A DISCUSSION OF SOME TECHNICAL ASPECTS OF SPEECH AIDS FOR POSTLARYNGECTOMIZED PATIENTS

HAROLD L. BARNEY (by invitation)

MURRAY HILL, N. J.

Since the development of the Western Electric Models 2A and 2B artificial larynges about thirty years ago,¹ there have been a number of developments in the general fields of acoustics and electronic circuitry which would be applicable to the design of an improved artificial larynx. A major development in electronic circuitry has been the invention and perfection of the transistor; this, coupled with miniaturization of circuit components such as acoustic transducers and batteries, has made possible radical improvements in hearing aids and other similar types of devices. Also improved acoustic and electronic circuit measurement techniques are now available using new instruments such as the sound spectrograph, for example. It would certainly be of interest to examine the possibilities afforded by these new techniques in the design of an artificial larynx. But before a choice can be made of a particular method of operation of an artificial larynx, there are a number of acoustic and electronic circuit aspects of the problem to be considered. It will be the purpose of this paper to examine some of these aspects in detail.

DESIGN OBJECTIVES FROM THE VIEWPOINT OF THE PATIENT

An exhaustive survey of opinions of postlaryngectomized patients has not been made to determine what would be an ideal type of artificial larynx, but the consensus of those patients and doctors who have been consulted is that an ideal device should have the following attributes:

1. Output speech volume equal to that of a normal speaker.
2. Output speech quality and pitch inflection like that of normal speech.

3. Unobtrusive; without visible wires, tubes, or other appurtenances, and small in size.
4. Reliable; with trouble-free operation for long periods of time.
5. Hygienically acceptable to the user.
6. Inexpensive price and low operating cost.

In addition, the device should be simple to operate so that a minimum of training is required. A design which attempts to satisfy all the above objectives must necessarily involve some compromises, if for no other reason than that an uncompromising compliance with the first four objectives would certainly require a design that would not be inexpensive.

In cases where good proficiency with esophageal speech can be acquired by the patient, this provides a solution to the problem that is generally acceptable, although the speech volume is usually weaker and the speech quality is considerably inferior to that of normal speech. However, there is a sizeable fraction, about a third, of all laryngectomized patients who cannot master esophageal speech for one reason or another, according to O. W. Lueders.² For this group an improved design of artificial larynx is needed.

The first design objective listed above, namely, that of a normal speech output volume, is a relatively easy one to meet. Normal conversational speech requires a total acoustic output of only about 20 microwatts when integrated over an interval of several seconds or more. The short term peak factors are such that on the peaks of strong syllables in conversational speech, the acoustic power output may be as much as 1,000 microwatts. Acoustic powers of this magnitude are quite easily radiated with small transducers, and various types of artificial larynges could be designed which would meet this requirement.

The second objective, of obtaining speech quality like that of normal speech, will be somewhat more difficult of attainment. To sound natural, speech should have pitch inflection, should have both voiced and unvoiced types of output, and should have spectra of energy distributions versus frequency that are rather carefully controlled according to the various speech sounds that are intended. These aspects will be discussed in more detail later in this paper.

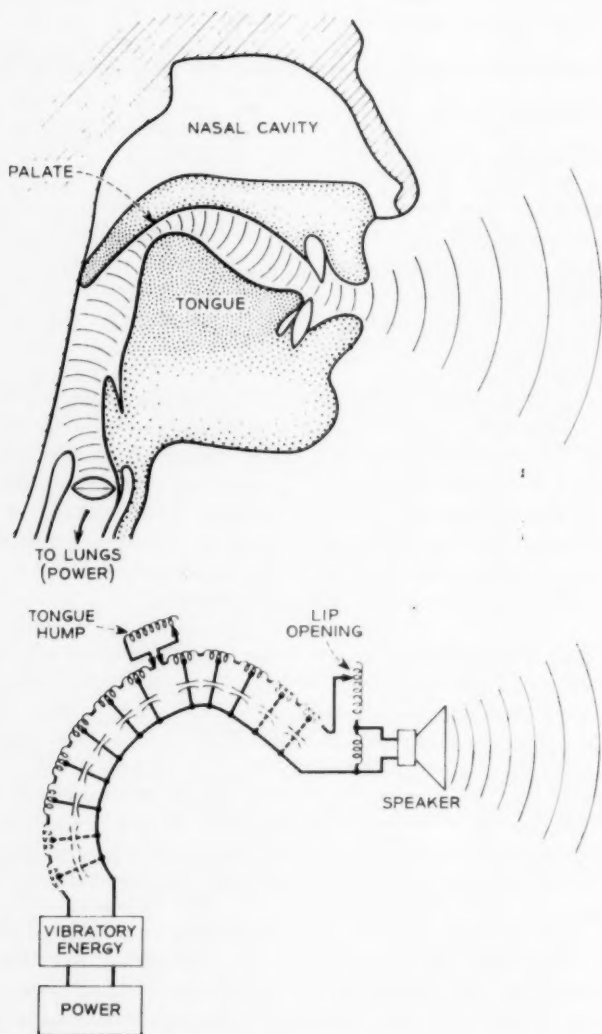


Fig. 1.—Sagittal section of the head showing vocal tract cavities and points of constriction. Lower part of the figure shows an electrical circuit analogue of the vocal tract.

The third objective, of making the device unobtrusive, is considered by all to be highly desirable. It is in this respect that the Western Electric Model 2 device is felt to be seriously deficient. Many postlaryngectomized patients express the view that the insertion of the tube into the mouth and the connection at the throat constitute sources of embarrassment. Judging from the efforts of hearing aid manufacturers to produce unobtrusive hearing aids, this desire to avoid the appearance of anything that would call attention to an infirmity is one to which serious attention should be given.

The fourth, fifth and sixth objectives of reliability, acceptability from a hygienic standpoint, and low cost are also obviously desirable. For an electronic device, the battery power consumption should be kept to a minimum in order to reduce costs of battery replacement. The manufacturing cost can be minimized by the use of commercially available transducers, transistors, and other component parts. In this respect, the Western Electric Model 2 device has been an expensive one to manufacture because of the special reed mechanism that has to be machined to close tolerances especially for it.

ACOUSTIC FACTORS WHICH AFFECT SPEECH QUALITY OF AN ARTIFICIAL LARYNX

In the normal production of voiced speech sounds, the initial source of sound is provided by the vocal cords. This source produces a complex sound which is rich in harmonic content. The quality of this sound is modified by the resonating action of the cavities of the pharynx, mouth and nose, and by constrictions at the back of the tongue and at the lips and teeth. The different combinations of the shapes of these cavities and constrictions give rise to the various voiced sounds of speech. The upper part of Figure 1 shows a sagittal section of the head and illustrates these cavities and constrictions. The lower part of Figure 1 shows an electrical circuit analogue of the vocal tract developed by H. K. Dunn, which has been found useful in studies of speech processes.³

As an illustration of the way in which the harmonic content of the speech wave is modified by a simple change in the vocal tract, consider the sound spectrogram of the sounds "ah" and "ee" shown in Figure 2. In going from "ah" to "ee," the only change in the vocal tract is the movement of the tongue hump from a back to a

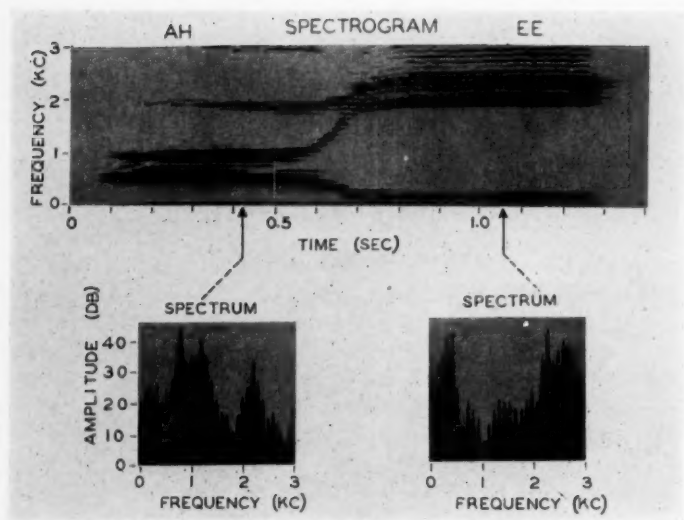


Fig. 2.—Sound spectrogram and spectra of the vowel sounds, "ah" and "ee," as spoken by a normal male talker.

front position in the mouth. The patterns of Figure 2 present an analysis of the sounds by visible speech techniques developed at Bell Telephone Laboratories by Potter, Kopp, and Green.⁴ In this spectrogram, time is portrayed on the horizontal scale from left to right, frequency on the vertical scale, and the intensity of a component of the sound at any time and frequency position is indicated by the darkness of the marking on the pattern at that time and frequency. This is a narrow band spectrogram, and the individual harmonics of the speech sound appear as closely spaced horizontal lines whose changing intensities determine the over-all pattern. Individual spectra may be portrayed at any particular time in the sound; for instance, spectra taken in the middle of the "ah" and "ee" sounds are also shown on Figure 2. In these spectra, frequency is indicated by the horizontal scale, and amplitude by the vertical scale. The individual spikes in the spectra are the fundamental and the harmonics of the sound, and the regions in which the cavity resonances of the vocal tract result in groups of harmonics being enhanced in amplitude are

called formants. These formant positions along the frequency scale are distinguishing characteristics of the various voiced sounds, and one requirement of any artificial larynx which produces natural sounding speech is that it be capable of producing formant patterns resembling those of normal speech sounds.

When the source of sound is introduced into the mouth cavity, as it is with the Western Electric Model 2 artificial larynx, the pharynx and nasal cavities do not have the same resonating effect to shape the spectrum of the output speech as they would if the sound were introduced at the glottis; consequently the formant patterns of some sounds cannot be made satisfactorily. This causes a loss of naturalness and intelligibility.

In order to study the effect of inserting the sound source at different points in the vocal tract, a standard ring armature receiver unit such as is used in telephone handsets, was equipped with a conical throat and a small flexible tube, as shown in Figure 3. With the tube as shown, the sound generated by the receiver could be inserted in the mouth. With the same tube, having the end inserted through the nasal cavity, down through the pharynx to a point just above the glottis, the sound could be inserted in a location to simulate the normal production of speech.

In order to simulate normal speech quality as closely as possible, considerable attention was given to getting a satisfactory sound source spectrum. For this purpose, a spectrum was desired which had the harmonic amplitudes approximately inversely proportional to the 1.5 power of the harmonic number. Although the spectrum of the volume velocity for normal vocal cords varies somewhat with the intensity of the cord tone produced, and also varies to some extent from one individual to another, the 1.5 power law is representative of an average condition. The acoustic properties of transducers and the tubes used to convey the sound to the mouth or to the throat ordinarily cause pronounced peaks or valleys in the spectrum of the sound source, unless careful attention is given to these factors in the design. A representative spectrum of the sound source with plastic tube is shown in Figure 4. This departs somewhat from the 1.5 power law, but is of the right general slope, and is free enough from pronounced peaks or valleys to be quite satisfactory.

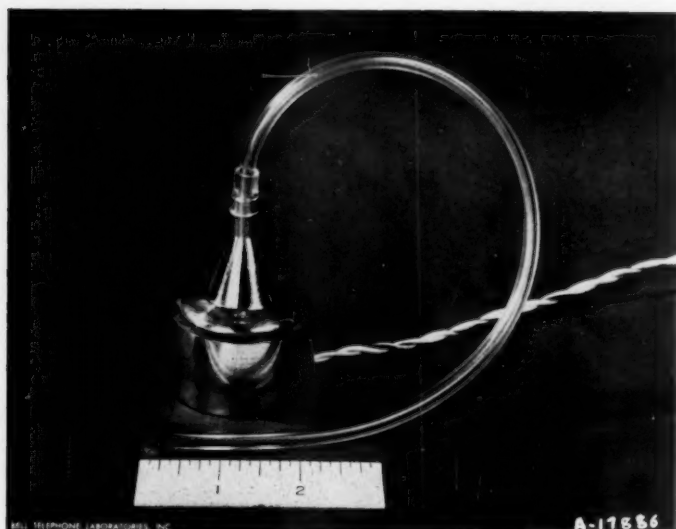


Fig. 3.—Special transducer for producing buzz tones, and a plastic tube for insertion of sound in pharynx or mouth.

With the sound source inserted near the vocal cords, brief listening tests showed that speech had a more natural quality than when the source was inserted in the mouth. This result confirmed the thesis that the most natural sounding speech would be that in which all of the vocal tract would be effective in shaping the output spectrum. Examples of sound spectra for a vowel sound spoken normally by a male speaker, then with the sound source in the pharynx and finally with the source in the mouth of the same male talker, are shown in Figure 5. This shows the spectra of the "oo" sound as in "book" for the three conditions. Inspection of these patterns shows that for the normal and artificial speech with the source in the throat, the four principal formants have nearly identical frequency positions. Their relative amplitudes, while different, are not so far out of line as to appreciably change the phonetic value of the sound. With the source in the mouth, however, the second formant is considerably more prominent than any of the others, the third

formant is at a higher frequency than in the other two conditions, and the fourth formant is completely missing. The presence of the tube in the mouth, preventing the tongue from assuming the appropriate position to form the vowel, plus the difference in effect of the vocal tract cavities in shaping the resonances which control the formant frequency positions and amplitudes, combine to give the result shown in Figure 5.

The effect of introducing the sound source in the mouth rather than at the glottis was also studied with the aid of the electrical analogue vocal tract which was shown on the lower part of Figure 1. In this circuit, the buzz tone was inserted in the line between the inductance simulating the tongue hump, and the output. This corresponded to insertion of the acoustic signal in the mouth, and the results were similar in that the vowel quality was appreciably inferior as judged by listening tests and by spectrographic analyses.

Since it appears that the preferred position for the sound source is in the pharynx, it is appropriate to consider the advantages and disadvantages of introducing the sound from the outside of the throat by means of a buzzer-like transducer. This has the advantage that has just been discussed, of using the entire vocal tract to modulate the source sound. However, it poses two obstacles to the attainment of satisfactory artificial speech. One of these is the large amount of power output required of the buzzer in order to get an acoustic signal through the flesh and cartilage into the pharynx. This is particularly true if a natural sounding speech output is desired, which requires a source spectrum having strong low frequency components. It is not too difficult to transmit acoustic energy into the flesh from a small transducer, if one is concerned only with components lying in the upper part of the audible frequency range. Such a source, having principally the higher frequencies in its output, would give a buzzy kind of quality rather than a normal sounding one. The acoustic problems attendant to radiating into the flesh, a broad band of frequencies extending from about 100 to several thousand cycles with a small transducer having a high efficiency, are too involved to be considered here, but they are formidable.

A second difficulty with an external transducer is the direct radiation of sound into the air, which, of course, is unmodulated

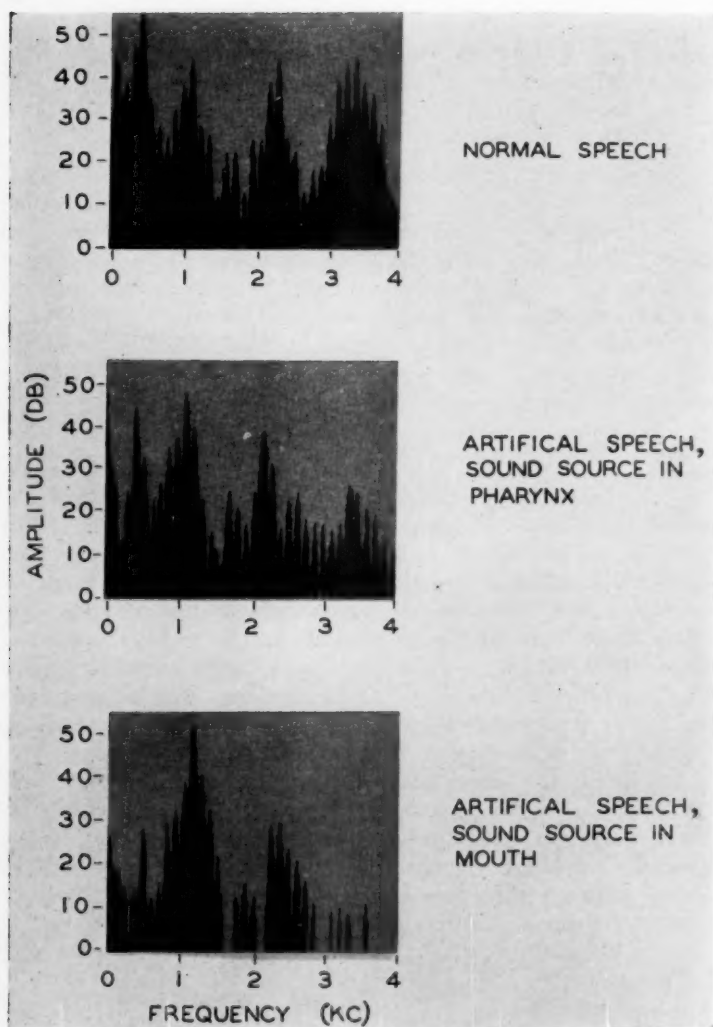


Fig. 5.—Sound spectra of vowel sound "oo" (as in "book") as spoken by a normal talker, and as produced artificially with the sound source in the pharynx, and in the mouth.

by the vocal tract, and therefore appears as a steady buzz accompanying the speech.

Inflection of the voiced sounds was mentioned previously as a factor in the naturalness of artificial speech. The degree of inflection need not be great, to obtain a considerable improvement in naturalness, as compared with monotone speech. Normal conversational speech contains inflections which correspond to a range of about an octave in the fundamental pitch frequency. A half octave range is quite sufficient for very acceptable inflections of speech. The inflection can be easily provided by a small rheostat in the appropriate part of an electronic driver circuit which actuates the transducer. For optimum effect, it should be operable in conjunction with the off-on button which starts the circuit buzzing. An arrangement of this sort for an electrical artificial larynx was developed by R. R. Riesz of Bell Telephone Laboratories at the time the original development work was being done on the Western Electric Model 2 artificial larynx.

PRODUCTION OF CONSONANT SOUNDS WITH ARTIFICIAL LARYNGES

Most of what has been discussed so far has dealt with the production of vowel sounds. The unvoiced sounds which are produced normally by turbulent air flow at some point in the vocal tract, can still be produced to some degree by the laryngectomized patient, even though there is no connection between the breath stream and the vocal tract. This is particularly so for plosive consonant sounds like "p," "t," and "k," which require only a small amount of air under pressure in the mouth to be released suddenly. Some of the fricative consonant sounds like "s," "f," and "th" (as in "thin") can be produced for short durations by forcing some of the air out of the mouth by tongue, jaw or cheek movements.

The Western Electric artificial larynx is well adapted to the production of these unvoiced sounds, particularly when they follow vowels in a word, since it provides a flow of air into the mouth cavity. Other types of artificial larynges, operated from electrical circuits, would not have this advantage and would have to depend on the method of forcing out impounded air from the mouth cavity, as described above.

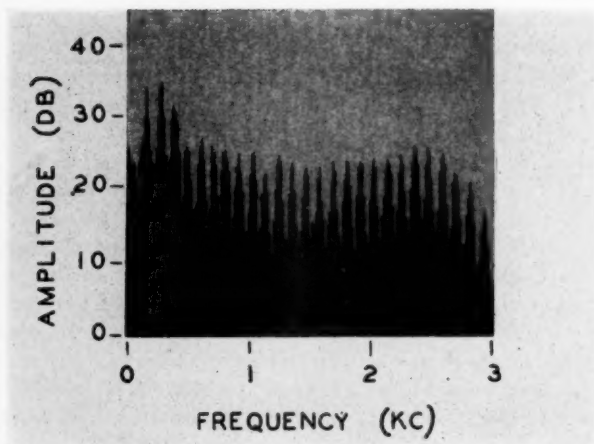


Fig. 4.—Sound spectrum of output of special transducer.

The intelligibility of consonant sounds is an important aspect of an artificial larynx, and various expedients have been considered for improving it. One idea that might be effective would be to provide two sources of sound energy, one the buzz for vowel sounds, and the other a hiss for sibilant or fricative consonants. If these were applied to the transducer alternatively under control of the talker, by a simple switch arrangement, improved naturalness and articulation might be obtained.

INTELLIGIBILITY OF ARTIFICIAL LARYNX SPEECH

In a study of artificial larynx and esophageal speech made at Ohio State University, Melvin Hyman⁵ measured the word articulation of a group of users of the Western Electric Model 2 artificial larynx, and found the average score to be about 48 per cent. In this study a multiple-choice intelligibility test list of phonetically balanced words was used. Corresponding scores on normal speech would be in the order of 98 per cent. Although the figure of 48 per cent may appear to be quite low, it is a characteristic of speech intelligibility that with a word articulation score of this value, discrete

sentence intelligibility will be in the order of 96 per cent. Hyman also found that the articulation scores with his group of esophageal speakers were not significantly different from the scores obtained with the Western Electric larynx, and that the quality of speech obtained with the artificial larynx was definitely preferred to that of esophageal speech by a large group of observers.

Some preliminary tests of word articulation with various types of artificial speech have been made at Bell Telephone Laboratories recently, in which it has been found that somewhat higher scores are made by expert users of the Western Electric device, and also by well-trained esophageal speakers. A very skilled user of the Western Electric larynx may have articulation scores as high as 70 per cent on multiple-choice phonetically balanced word lists. This would correspond to a discrete sentence intelligibility of about 99 per cent. It would seem that a desirable objective for a new artificial larynx design would be to obtain articulation scores of this order with a reasonably short period of training for the average user.

SUMMARY

To summarize, a number of aspects of the speech process have been considered, with the objective of shedding some light on the artificial larynx problem. It has been shown that the most suitable point to introduce the sound from a transducer is into the pharynx. Ideally, the best solution from an acoustic standpoint would be to use surgical means to place a small transducer so that its output could be applied to the pharyngeal cavity directly. If this is not advisable because of danger of possible infections or for other medical considerations, two possible alternatives are to conduct the sound into the mouth through a tube, as is done with the Western Electric Model 2 device, or by means of a vibrating driver applied to the outside of the throat. It is anticipated that both of these would fall somewhat short of the tentative design objectives enumerated at the beginning of this paper, in one or more respects. However, with proper acoustic and electronic circuit design, either should be capable of giving adequate intelligibility in the hands of the average user, and the addition of a conveniently operated pitch inflection control will provide a much more natural sounding speech.

BELL TELEPHONE LABORATORIES

REFERENCES

1. Riesz, R. R.: Description and Demonstration of an Artificial Larynx. *J. Acoust. Soc. Am.* 1:273-289, 1930.
2. Lueders, Oscar W.: Use of the Electrolarynx in Speech Rehabilitation. *A.M.A. Arch. of Otolaryng.* 63:133-134, 1956.
3. Dunn, H. K.: The Calculation of Vowel Resonances and an Electrical Vocal Tract. *J. Acoust. Soc. Am.* 22:740-753, 1950.
4. Potter, R. K., Kopp, G. A., and Green, H. C.: *Visible Speech*. D. Van Nostrand, New York, 1947.
5. Hyman, Melvin: An Experimental Study of Artificial Larynx and Esophageal Speech. *J. Speech and Hearing Disorders* 20:291-299, 1955.

Abstracts of Current Articles

EAR

Sudden Bilateral Hearing Impairment, Either of Simultaneous Onset or One Side Following the Onset of the Other One

Lehnhardt, E.: Zschr. Laryng. 37:1-16, 1958.

This is a report of 13 cases with varying degrees of impairment; simultaneous onset in only four of them; vestibular symptoms were absent in all cases. The (hypothetical) underlying pathology is discussed. In the author's experience, the cases are amenable to treatment if the hearing impairment is not too severe and treatment is begun early. The chances for recovery are poor in cases of complete deafness. The treatment is directed toward eradication of dental foci; elimination of disorders of the autonomic nervous system, of vascular disorders, and of allergic causes.

TONNDORF

Cytological Changes Within a Lymphnode Metastasis Under the Effect of X-ray Therapy

Schweitzer, L.: Medizinische, 1957, Nr 51, 1910-1911.

Puncture of a lymphnode with carcinomatous metastases was performed at regular intervals during the course of x-ray treatment. Cytological examination revealed the characteristic changes of carcinomatous cells under the effect of x-ray which are clearly differentiated from the reactions of normal cells. The author hopes, by employing this cytological follow-up method, to establish a clinical procedure for early diagnosis of carcinomatous metastases which is so important for the prognosis and management of patients with carcinomas.

TONNDORF

Modern, Non-laryngological Indications for Tracheotomy

Krabl, P.: Die Medizinische, 1958, Nr. 9, p. 339-343.

According to the author, the indications for tracheotomy have changed in recent years. While formerly the operation was performed mainly for the relief of respiratory difficulties in the upper air passages, today it is also performed in cases of incapacity in breathing and in swallowing due to diseases of the central nervous system: meningitis, encephalitis, poliomyelitis, exogenic and endogenic poisoning, etc. The tracheostoma is used in these cases to aid artificial respiration. The drainage of pharyngeal secretions into the air passages is prevented by an inflated rubber cuff around the canula and also by suction through the stoma. Intubation may be performed for the same purpose. However, its duration is limited to a few hours' time because of the danger of laryngeal injury. The author reports 18 cases which illustrate the indications for tracheotomy.

TONNDORF

Endolymphatic Oxygen Tension in the Cochlea of the Guinea Pig

Misrahy, G. A., Hildreth, K. M., Shinabarger, E. W., Clark, L. C., and Rice, E. A.: J. Acoust. Soc. Am. 30:247-250, 1958.

Many previous investigators have given indirect evidence to the fact that the function of the organ of Corti depends largely upon a sufficient supply of oxygen and also that this oxygen is most probably delivered by the stria vascularis reaching the organ of Corti by a simple process of diffusion. Now G. A. Misrahy and his co-workers have been able to measure quantitatively the oxygen tension within the scala media of the guinea pig. To this end, they improved an existing polarographic method using cochlear microelectrodes (the technical details of polarography are beyond the scope of this review). The system was calibrated so that it gave direct readings of O_2 tension in terms of mm Hg. Near the stria vascularis, O_2 tension was found to be in the order of 55-70 mm Hg (for comparison: for the arterial blood O_2 tension is generally given as 75-90 mm Hg). When the electrodes were moved deeper into the scala media, the values decreased gradually to 16-25 mm Hg, strongly suggesting that O_2 was supplied by the stria. O_2 tension was found to decrease with hypoxia and also with sound exposure indicating that the function of the organ of Corti is definitely aerobic. This ingenious and important work (in the reviewer's opinion) is currently continued by Dr. Misrahy.

TONNDORF

PHARYNX

The Indication and Technique of Surgical Decompression of the Recurrent Laryngeal Nerve

Miehlke, A.: Zschr. Laryng. 37:44-53, 1958.

Discussed are the various causes of paralysis of the vocal cords following thyroidectomy. Actual severance of the nerve is a rather infrequent cause. More frequently, the paralysis is caused by surgical trauma: overstretching of the nerve, damage from ligatures, or due to cicatricial compression. The optimal time for surgical revision and/or decompression is four to five months after thyroidectomy, according to available data on the speed of degeneration of peripheral nerves (and the recurrent nerve in particular). Surgical landmarks for the nerve are given and the technique of decompression is described.

TONNDORF

Books Received

Chemistry and Biology of Mucopolysaccharides

A symposium of the Ciba Foundation, edited by *G.E.W. Wolstenholme, O.B.E., M.A., M.B., B.Cb.* and *Maeve O'Connor, B.A.* Cloth, 8vo. xii - 323 pp., illustrations, tables and diagrams. Boston, Little, Brown and Co., 1958. Price \$8.50

The inquiring otolaryngologist, many of whose major problems are linked with the connective tissues, will find this book of absorbing interest. In spite of, or perhaps because of, our pre-occupation with cytology the intercellular ground substance has until recently remained a medical "blind spot." The inclusion of the subject as one of the themes of the recent international congress has stimulated the otolaryngologist and no less than three papers related to it will be found in this issue of the ANNALS.

The symposium deals with four basic topics: lipopolysaccharides, the chemistry and distribution of sialic acid, blood group substances and mucopolysaccharides in connective tissue. There are 18 papers and voluminous discussions, a fair part of which concern the otolaryngologist, provided that his training is such that he can understand them. In any case they deal with such elemental things as infection, resistance and allergy and are well worth plowing through.

Recent Advances in Otolaryngology

By *F. Boyes Korkis, M.B., Ch.B. (N.Z.), D.L.O. (Eng.), F.R.C.S. (Ed.), F.R.C.S. (Eng.)*, Surgeon and Dean, Metropolitan Ear, Nose and Throat Hospital; Senior Consultant Otolaryngologist, Hillingdon Hospital; Hon. Otolaryngologist, Royal Academy of Dramatic Art, etc. With a foreword by *R. Scott Stevenson, M.D., F.R.C.S. (Ed.)*. Cloth, 8vo. viii - 438 pp., 144 illustrations and 2 color plates. Boston, Little, Brown and Co., 1958. Price \$12.00

This is the third of a familiar series of the same name begun in 1935 by Scott Stevenson. The book is not in any sense a revision of its predecessors, but is strictly a review of the literature of the past decade. The author's personal reactions are expressed rather by his discrimination in selecting the papers reviewed than by direct criticism, and the field has been competently covered.

"I am glad," writes Stevenson in his foreword, "that Mr. Boyes Korkis has been able to resist the temptation to farm out various

chapters to other colleagues, as is so general in books on otolaryngology," and we agree that this single authorship adds much to the prestige of the book.

Clinical Enzymology

By six authors, edited by *Gustav J. Martin, Sc.D.*, Research Director, The National Drug Co., Philadelphia. Cloth, large 8vo. 241 pp. Boston, Little, Brown and Co., 1958. Price \$6.00

A full discussion of the chemistry, physical chemistry, and the biochemical and pharmacological characteristics of enzymes which have been used clinically, together with the rationale of enzyme therapy.

Rehabilitation of the Cardiovascular Patient

By *Paul Dudley White, M.D.*, Massachusetts General Hospital, Boston; *Howard A. Rusk, M.D.*, Professor and Chairman, Department of Physical Medicine and Rehabilitation, New York University-Bellevue Medical Center, New York; *Philip R. Lee, M.D.*, Department of Internal Medicine, Palo Alto Clinic, Palo Alto, California; and *Bryan Williams, M.D.*, Clinical Instructor in Medicine, University of Texas Southwestern Medical School, Dallas. Cloth, 8vo. xiv + 176 pp., 31 illustrations. New York, McGraw-Hill Book Company, Inc., 1958. Price \$7.00

A manual directed to all physicians having to do with rehabilitation, but dealing specifically with the practical day-to-day management of patients with vascular incapacities.

Sprachhörprüfmethode (Methods in Speech Audiology Testing)

By *Prof. Dr. Med. Dipl.-Ing. Kurt Schubert*, Universitäts-Hals-Nasen-Ohrenklinik, Bonn, Germany. Cloth, large 8vo. 361 pp., 187 illustrations. Stuttgart, Georg Thieme Verlag, 1958 (in U.S. Intercontinental Medical Book Corporation) Price \$16.45. (In German)

This is a thorough-going monograph on the subject, dealing with all its phases and well illustrated with tables and graphs. Being in German, and dealing with German language, it will have a limited usefulness for American audiologists. It is part of a series on Industry and Health.

Yearbook of Ear, Nose and Throat, and Maxillofacial Surgery

Edited by *John R. Lindsay, M.D.*, Professor and Head of the Section of Otolaryngology, University of Chicago School of Medicine; *Dean M. Lierle, M.D.*, Professor and Head of the Department of Otolaryngology and Maxillofacial Surgery, State University of Iowa College of Medicine; and *William C. Huffman, M.D.*, Professor of Otolaryngology and Maxillofacial Surgery, State University of Iowa College of Medicine. Cloth, 8vo. 383 pp., 96 illustrations. Chicago, The Year Book Publishers, Inc., 1958.

As indicated above, the present volume of this classic series dissolves the long partnership between otolaryngology and ophthalmology and takes on a newcomer: maxillofacial surgery. The year's literature is reviewed with the usual competence; there are only occasional editorial comments. It is a commentary on fashions in medical thinking to discover that of 368 pages of reviews only 15 deal with the nose. As of today the ears have it.

Surgery in World War II (Ophthalmology and Otolaryngology)

Editor-in-Chief, Colonel John Boyd Coates, Jr. MC; Editor for Ophthalmology, M. Elliott Randolph, M.D.; Editor for Otolaryngology, Norton Canfield, M.D.; Associate Editor, Elizabeth M. McFetridge, M.A. With 24 contributors. Cloth, xxiii - 605 pp., illustrated, Office of the Surgeon General, Department of the Army, Washington, D.C., 1957.

Of interest to every medical officer, past and present, in any war is this impressive definitive volume just issued by the Surgeon General of the Army.

The work of a score of contributors, it deals with every phase of military ophthalmology and otolaryngology from training and materiel to the organization of hospitals and the handling of large numbers of patients under general and special conditions. There are detailed studies of types of diseases and injuries encountered in War II with statistics and emphasis on after-treatment and rehabilitation.

This is an invaluable text for medical students who have not yet entered their term of military service and especially those who contemplate military careers.

Notices

A LETTER TO THE EDITOR

HEARING LEVEL, HEARING LOSS, AND THRESHOLD SHIFT

The following excerpt from our recent paper in the AMA Archives of Industrial Health entitled, "The Medical Principles of Monitoring Audiometry," speaks for itself (H. Davis, Hoople, G. and Parrack, H. O.: The Medical Principles of Monitoring Audiometry. A.M.A. Arch. Indus. Health 17:1-20, 1958):

HEARING LEVEL, HEARING LOSS, AND THRESHOLD SHIFT

The familiar term "hearing loss" includes three quite distinct concepts, and much confusion and many unnecessary arguments have occurred in court rooms, in committee rooms and in clinics because of this semantic monstrosity. These three concepts are 1) the otological meaning: A *symptom* indicating an *abnormal condition* of hearing, 2) the audiological meaning: the *status of hearing* as measured by a reading in decibels on the hearing-loss dial of an audiometer, and 3) the common sense meaning: *a change for the worse* in the sensitivity of hearing. It has been particularly difficult in medico-legal situations to reconcile the second meaning with the other two and to explain that the normal range of hearing extends from a hearing loss of minus ten to a hearing loss of plus ten or fifteen decibels. It is hard to convince a jury that a man can have a hearing loss of ten decibels when his hearing may actually never have *changed* at all. It has been very difficult and cumbersome, also, in our own writing to distinguish clearly between the *status* of hearing, to which disability of hearing and physical standards for military duty, etc., are related, and a *change in status* of hearing, which is the central concept of monitoring audiometry.

We shall use, and we recommend for general usage, three different terms for three different, although related, meanings of "hearing loss."

1. "Hearing level" is the deviation in decibels of an individual's threshold of hearing from the American Standard value for the refer-

ence zero for audiometers. This is a measure of the *status of hearing*. It is read directly on the "hearing loss" scale of an audiometer. To be completely explicit we should say "threshold hearing level," meaning "the number of decibels above the sound pressure level that corresponds to the reference threshold of hearing." In general, however, the shorter phrase "hearing level" should be adequate.

The military physical standards of acceptable hearing and some of the "monitor limits" that are to be described later are now stated in terms of "hearing loss," but in accordance with the new usage here proposed they should be stated in terms of "hearing level."

"Hearing level" refers to a single measurement made with a standardized instrument and referred to an arbitrary zero reference level. Larger numbers indicate that more acoustic energy is needed to reach the threshold of hearing. The term is analogous to the physicist's "sound pressure level." Both specify a level relative to a reference level, but the reference for hearing is different at different frequencies.

The term "*speech hearing level*" or "hearing level for speech" is a convenient designation for the average of the hearing levels for the frequencies necessary for the understanding of speech.

2. "*Hearing loss*" will only be used to refer to the *symptom* of reduced auditory sensitivity, as in the phrases "conductive hearing loss," "noise-induced hearing loss," etc. These examples illustrate the otologist's automatic concern with the question of the mechanism of a hearing loss, its cause, and its possible cure. "Hearing loss" for the otologist is the modern substitute for the old phrase "partial deafness" and means almost exactly the same thing as "auditory impairment." It may also be used in a general way to refer to the *process* of losing auditory sensitivity, as in the phrase, "he suffered a hearing loss."

3. A "*threshold shift*" is the deviation in decibels from an individual's own previously established reference audiogram. It is a *measure of change in the status of hearing*. It is the term we shall usually employ instead of the more familiar "loss of hearing." Unless otherwise specified it is assumed that the shift of threshold is upward to a higher hearing level, meaning that the change in hearing is a change for the worse. A change for the better will usually be termed a "recovery." It is very useful to distinguish "temporary threshold shift" from "persistent threshold shift."

We particularly recommend the use of the term, "hearing level."

Hallowell Davis, Central Institute for the Deaf,
St. Louis, Missouri

Gordon D. Hoople, 1100 East Genesee Street,
Syracuse, New York

Horace O. Parrack, Wright Air Development Center,
Dayton, Ohio

SOCIETE FRANCAISE d'OTO-RHINO
LARYNGOLOGIE

The Societe Francaise d'Oto-Rhino-Laryngologie will hold its annual congress in the grand Amphithéâtre de la Faculté de Médecine de Paris, rue de l'Ecole de Médecine, October 20-23, 1958.

The official topics for discussion will be:

- 1) Traitement du Cancer de las base de la langue.
Mm. Huet, Gignoux, Berard, Andre et Labayle.
- 2) La Cortisone et l'A.C.T.H. en O.R.L.
Mm. Aubin, Terracol et Guerrier.

For information please address the Secretary General, Dr. H. Guillon, 6, avenue Mac-Mahon, Paris.

AMERICAN LARYNGOLOGICAL ASSOCIATION

Copies of the Transactions of the American Laryngological Association are available for general distribution at \$8.00 a copy. Please send request with check to Dr. Edwin N. Broyles, Editor, Transactions, 1100 North Charles St., Baltimore 1, Md.

SEVENTH INTERNATIONAL CONGRESS
OF BRONCHESOPHAGOLOGY

The Seventh International Congress of Bronchoesophagology will take place September 12 - 14, 1958, at Kyoto, Japan. The summary of papers, within 800 words, should be sent to the director not later than the end of April, 1958.

Director: Prof. Mituharu Goto, M.D.
Otorhinolaryngological Clinic
Kyoto University Hospital
Kyoto, Japan

AMERICAN ACADEMY OF
OPHTHALMOLOGY AND OTOLARYNGOLOGY
HOME STUDY COURSES

The 1958-1959 Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1 and continue for a period of ten months. Detailed information and application forms may be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 15 Second Street, S.W., Rochester, Minnesota. Registrations should be completed before August 15.

BÁRÁNY MEDAL

The Bárány Jubilee Medal for 1958 has been awarded by the Medical Faculty of Uppsala University to C. S. Hallpike of London for his experimental and clinical investigations of the functions of the vestibular system and of optomotorial nystagmus.

THIRD INTERNATIONAL CONGRESS OF ALLERGOLOGY

The Third International Congress of Allergology will take place at Paris, France, October 19-26, 1958. It is sponsored by the International Association of Allergology and the French Allergy Association.

For registration and Congress information please address Dr. B. N. Halpern, 197 Boulevard St. Germain, Paris VII, France.

For travel and post-convention tours consult Thos. Cook and Son, 166 N. Michigan Blvd., Chicago, Ill.

UNIVERSITY OF ILLINOIS

The University of Illinois College of Medicine Department of Otolaryngology announces its Annual Otolaryngologic Assembly from September 29 through October 5, 1958. The Assembly will consist of an intensive series of lectures and panels concerning advancements in otolaryngology, and evening sessions devoted to surgical anatomy of the head and neck and histopathology of the ear, nose and throat.

Interested physicians should write direct to the Department of Otolaryngology, 1853 West Polk Street, Chicago 12, Illinois.

The next postgraduate course in Laryngology and Bronchoesophagology to be given by the University of Illinois College of Medicine is scheduled for the period October 27 through November 8, 1958. The course is under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

TEMPLE UNIVERSITY

There will be a postgraduate course in Bronchoesophagology November 3 to 14, 1958, and a postgraduate course in Laryngology and Laryngeal Surgery December 1 to 12, 1958, given in the Department of Laryngology and Bronchoesophagology, under the direction of Drs. Chevalier L. Jackson and Charles M. Norris.

The tuition fee for each course is \$250. Further information can be obtained from Dr. Chevalier L. Jackson, 3401 N. Broad Street, Philadelphia 40, Penna.

AMERICAN RHINOLOGIC SOCIETY

The Annual Program Meeting of the American Rhinologic Society will be held at the Palmer House, Chicago, on Friday, October 17, and Saturday, October 18, 1958.

The meeting will start at 1:30 p.m. on October 17 and adjourn at 4:00 p.m. on Saturday, October 18, 1958.

AMERICAN ASSOCIATION FOR CLEFT PALATE
REHABILITATION

The American Association for Cleft Palate Rehabilitation will hold its 17th Annual Convention at the Sheraton Hotel in Philadelphia on Thursday, Friday and Saturday, April 30, May 1 and 2, 1959.

This Association is composed of medical, dental and paramedical specialists interested in the subject.

ANNALS

The ANNALS wishes to repurchase copies of the March 1955 and the March 1957 issues, which are out of print, at two dollars a copy. These should be delivered to Manager of the ANNALS, P. O. Box 1345, Central Station, St. Louis 1, Mo.

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Meeting: The Homestead, Hot Springs, Va., March 10 and 11, 1959

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Meeting: The Homestead, Hot Springs, Va., March 8 and 9, 1959

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

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Meeting: The Homestead, Hot Springs, Va., March 10, 11, 12, 1959

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY

Chairman: Victor R. Alfaro, M.D., Washington, D.C.

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Meeting: Atlantic City, June 8-12, 1959

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Meeting: The Homestead, Hot Springs, Va., March 13 and 14, 1959

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President: Joseph W. Hampsey, M.D., Grant Bldg., Pittsburgh 19, Pa.

Secretary-Treasurer: Daniel S. DeStio, M.D., 121 S. Highland Ave., Pittsburgh 6, Pa.

Meeting: Palmer House, Chicago, Illinois, October 16 and 17, 1958

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: H. Leroy Goss, M.D., 620 Cobb Building, Seattle 1, Wash.

Secretary-Treasurer: Homer E. Smith, M.D., 508 East South Temple, Salt Lake City, Utah

THE SOCIETY OF MILITARY OTOLARYNGOLOGISTS

President: Captain William C. Livingood, United States Navy (MC)

Secretary-Treasurer: Lt. Colonel Stanley H. Bear, United States Air Force (MC)

CANADIAN OTOLARYNGOLOGICAL SOCIETY

President: Dr. G.M.T. Hazen, 208 Canada Bldg., Saskatoon, Sask.

Secretary: Dr. G. A. Henry, Medical Arts Bldg., Suite 328, Toronto, Ont.

INTERNATIONAL BRONCHESOPHAGOLOGICAL SOCIETY

President: Dr. Jo Ono, Tokyo

Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa.

Meeting: Kyoto, Japan, Sept. 12-14, 1958

PAN-AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY

President: Dr. Jose Gros, Havana

Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa.

Meeting: Sixth Panamerican Congress, Brazil, August 12, 1958

